Psychological Monographs

EDITED BY

JOSEPH PETERSON, GEORGE PEABODY COLLEGE

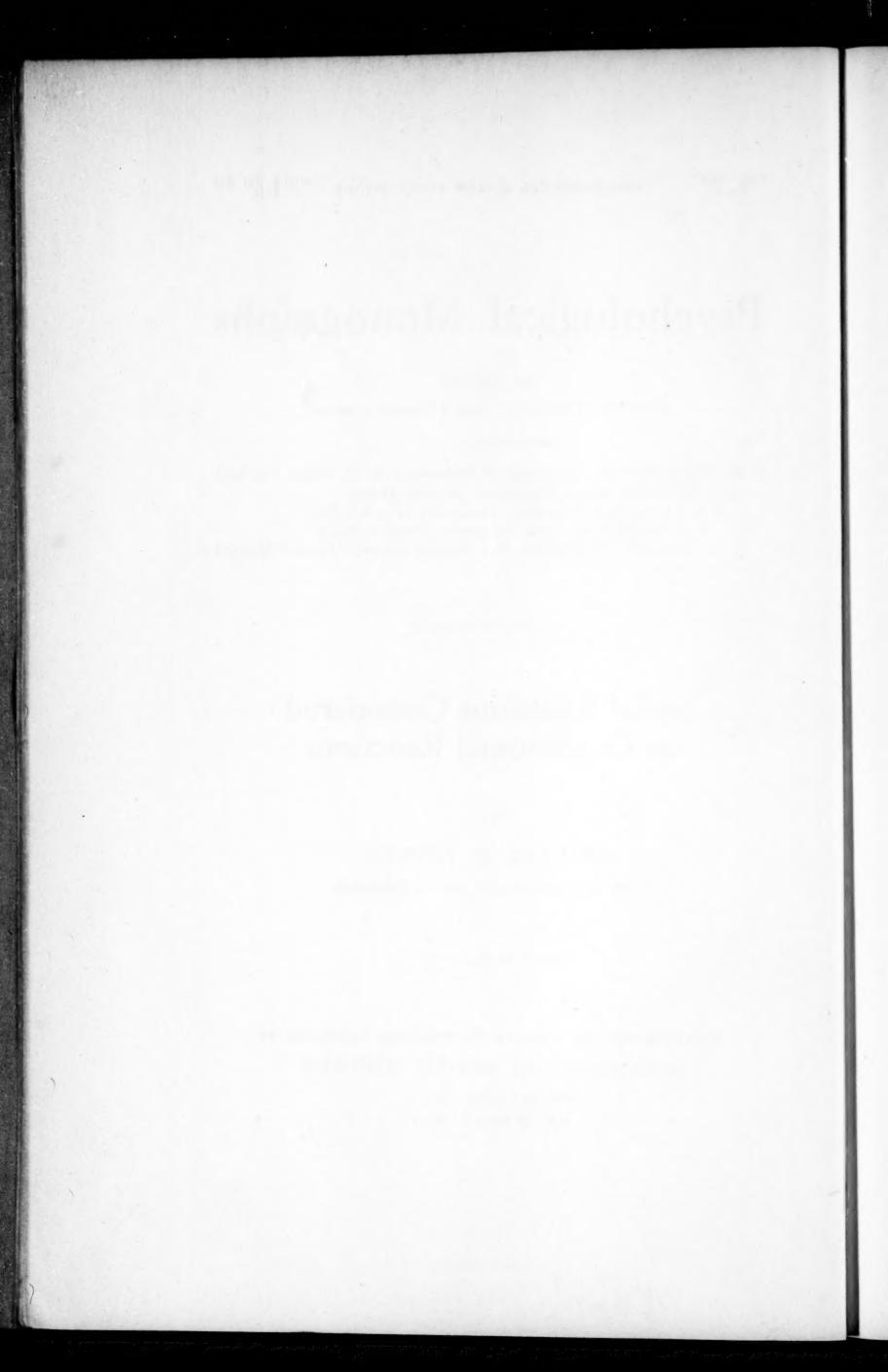
S. W. FERNBERGER, University of Pennsylvania (J. Exper. Psychol.)
W. S. HUNTER, Clark University (Psychol. Index)
H. S. LANGFELD, Princeton University (Psychol. Rev.)
E. S. ROBINSON, Yale University (Psychol. Bull.)
JOSEPH PETERSON, Geo. Peabody College (Psychol. Monog.)

Serial Reactions Considered as Conditioned Reactions

BY

WILLIAM M. LEPLEY
THE PENNSYLVANIA STATE COLLEGE

PUBLISHED FOR THE AMERICAN PSYCHOLOGICAL ASSOCIATION BY
PSYCHOLOGICAL REVIEW COMPANY
PRINCETON, N. J.
AND ALBANY, N. Y.



FOREWORD

The author of this study is indebted to certain persons for their various helpful influences. First among these should be mentioned the several members of the Department of Psychology of The Pennsylvania State College. In addition, the author wishes to acknowledge a most valuable remote association with Professor Clark L. Hull of Yale University. These kindly advisors have contributed encouragement and critical guidance admittedly beyond objective measurement.

WILLIAM M. LEPLEY

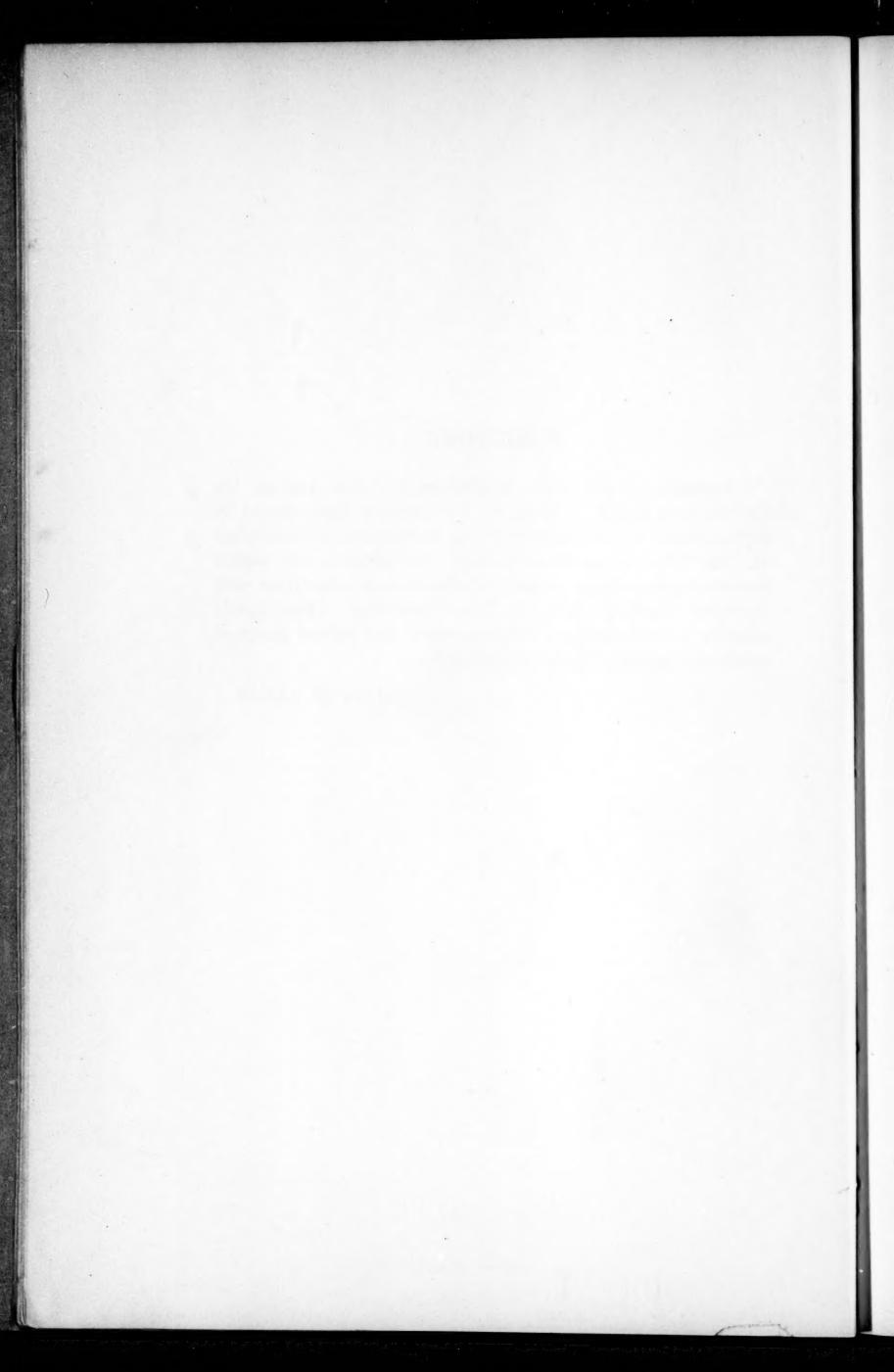
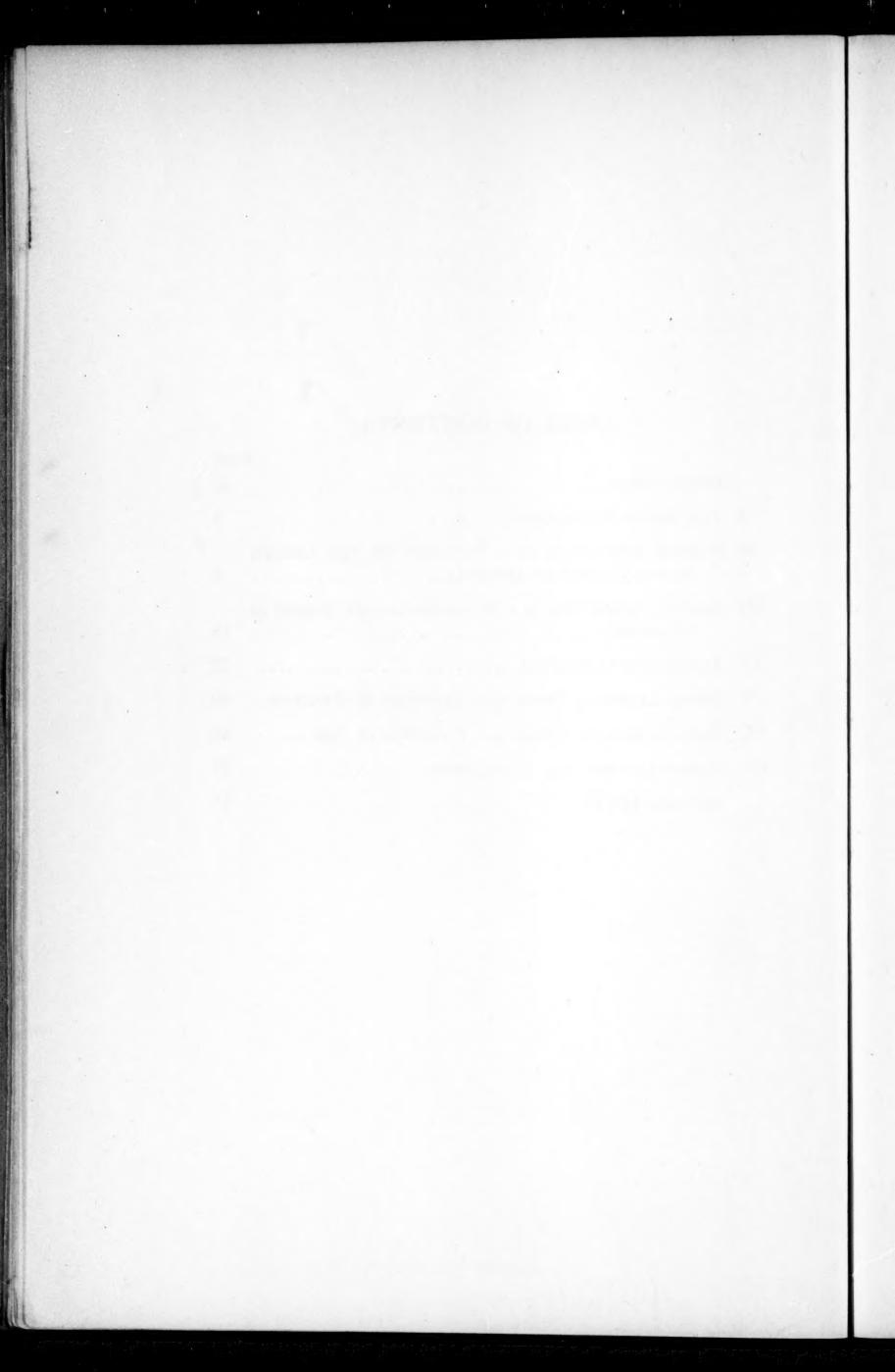


TABLE OF CONTENTS

	P	AGE
	Introduction	vii
I.	THE MAJOR HYPOTHESIS	3
II.	REMOTE EXCITATION AS A FUNCTION OF THE LENGTH OF THE FORGETTING INTERVAL	6
III.	REMOTE EXCITATION AS A FUNCTION OF THE DEGREE OF LEARNING	18
IV.	Associative Inhibition	28
V.	SERIAL LEARNING ORDER AS A FUNCTION OF PRACTICE	40
VI.	SERIAL LEARNING ORDER AS A FUNCTION OF AGE	46
VII.	RECAPITULATION AND CONCLUSION	52
	Bibliography	55

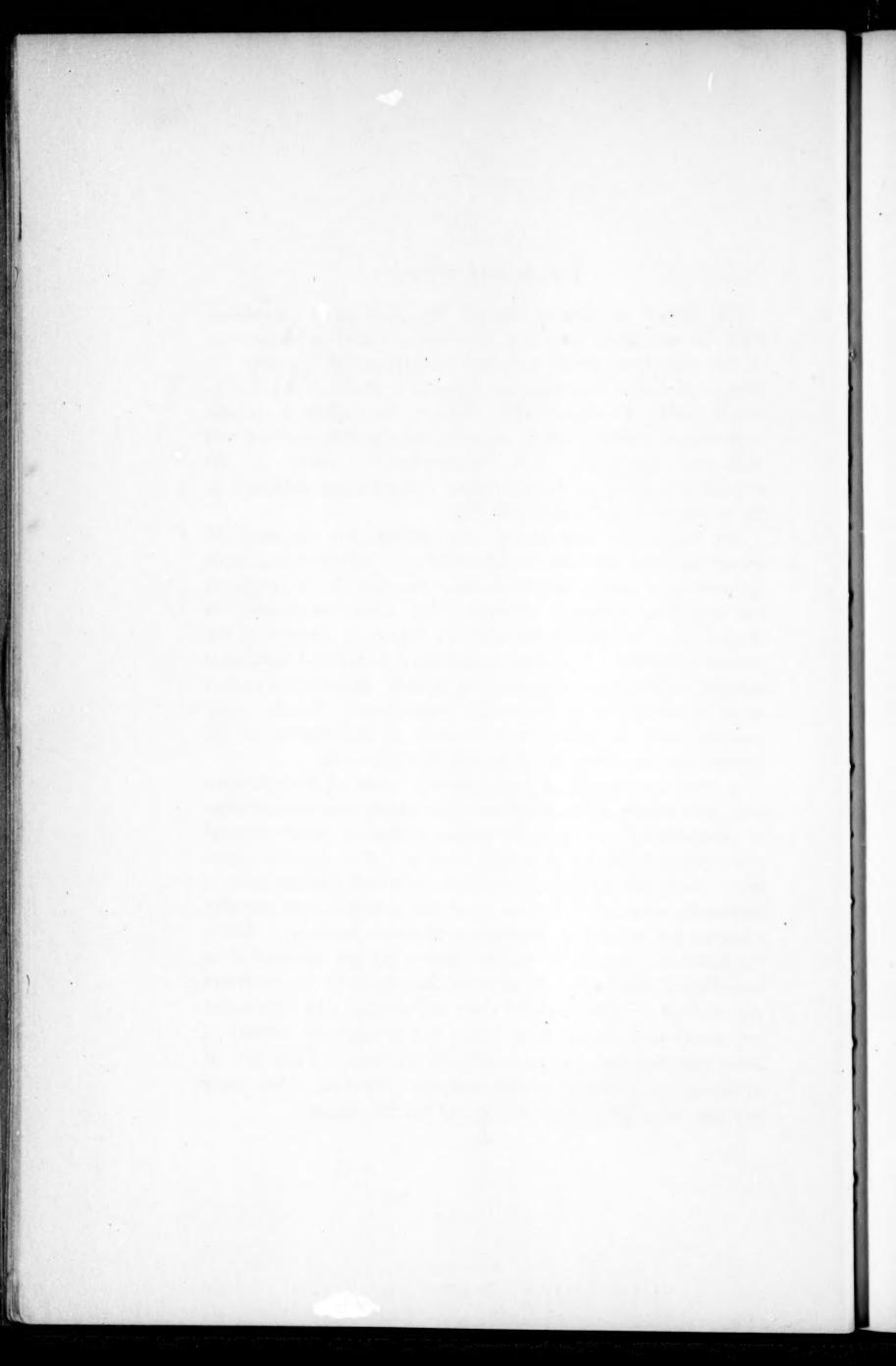


INTRODUCTION

The author of this monograph has two major objectives. First, he intends to present a reformulation and an elaboration of the hypothesis which appeared recently under the title: A Theory of Serial Learning and Forgetting Based Upon Conditioned Reflex Principles (19). Second, he intends to submit experimental evidence which seems to support this modified and elaborated hypothesis. The reformulation consists of the detailed exposition of the alternative interpretation suggested in the above-mentioned article (19, 283).

The two major experiments were planned for the study of remote associations under varied conditions. An attempt is made to investigate remote association as a function of the length of the forgetting interval. Further, this same phenomenon is studied as a function of the subject's degree of mastery of the learned materials. The minor experiments, concerned with serial learning order, were suggested by certain casual observations made in the course of the major experiments. Briefly, serial learning order is studied as a function of the practice of the learner, and as a function of the learner.

In order to forestall, at least partially, some of the timeworn criticisms usually directed against stimulus-response formulations of psychological problems, the author wishes to declare himself with respect to the use of certain terms and other symbolic practices. First, the selection of stimulus units and reaction units is admittedly arbitrary. Second, it is not assumed that complex reactions are built by the summation of simple reactions. Third, the illustrative figures in the first chapter are not intended to be neurological diagrams. In general, the simplicity of treatment was evolved in the interest of clear exposition. The hypotheses are intentionally stated in an exact and unequivocal manner in order that they may be examined with precision. Their lack of ambiguity is a matter of the author's intention. They were intended for experimental testing and not for debate.



CHAPTER I

THE MAJOR HYPOTHESIS 1

This theoretical development will be cast in such terms as will best harmonize with the experimental procedure to follow. We shall be dealing with serial material and we shall represent the members of these series by the conventional symbols wherever possible. Further, we shall let the stimulus symbols represent visually presented nonsense syllables. Below is given a key to the interpretation of the diagrams.

S	Visual stimulus
R	Vocal response
S	Kinaesthetic and auditory stimuli arising from R
>	Produces or gives rise to
>	Previously established language habits
>	Immediate excitatory tendencies
>	Remote excitatory tendencies
	Inhibitory tendencies

In the learning of serial acts it is assumed:

- 1. That immediate associations or immediate excitatory tendencies are established having the nature of higher order, simultaneous, or near simultaneous conditioned reactions.
- 2. That remote excitatory tendencies, as demonstrated by Ebbinghaus (6) and by Hall (8) are established, having the dual nature of trace and delayed conditioned reactions, as demonstrated by Pavlov (27, 88–105, 39–40).

We shall elaborate these assumptions in order that they may be made precisely meaningful, and that their implications may be clearly indicated. We shall follow the hypothetical establish-

¹ This chapter contains elaborated excerpts from the author's previous article, "A Theory of Serial Learning and Forgetting Based upon Conditioned Reflex Principles," which was published in the May, 1932, issue of the *Psychological Review*. These elaborated excerpts and the five figures in this chapter are included in this writing by permission of the Psychological Review Company.

ment of tendencies or associations throughout the learning of a series of nonsense syllables barely to be learned, that is, to be learned to the point of one perfect anticipatory performance. First, we have, at the beginning of practice, a situation wherein the subject simply responds vocally to the successively presented members of the series. This situation is represented diagrammatically in Fig. 1. The visual stimuli S_1 , S_2 , etc., evoke respectively.

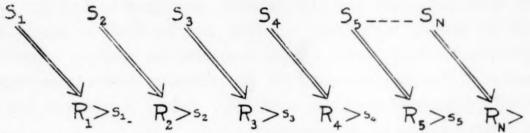


Fig. 1. Visual stimulus series, vocal response series and the auditory-kinaesthetic stimulus series arising from the vocal response series.

tively the vocal responses R₁, R₂, etc. These responses are, of course, made possible by previously established language habits.

It is to be noted that s_1 (kinaesthetic and auditory) arising from R_1 , and also S_1 (visual) immediately precede $S_2 = R_2$. This relationship suggests that, following the redintegrative principle, the complex $\frac{S_1}{s_1}$ may acquire the ability to evoke R_2 without S_2 being present. In other words, $\frac{S_1}{s_1}$ becomes a conditioned stimulus for response R_2 and $\frac{S_1}{s_1} = --->R_2$ becomes a simultaneous or near simultaneous conditioned reaction. Likewise, $\frac{S_2}{s_2}$ and $\frac{S_3}{s_3}$ become the conditioned stimuli for R_3 and R_4 respectively, and so on throughout the series. Thus, our subject, being stimulated by S_1 and having responded R_1 , may, being stimulated by S_1 , auditory-kinaesthetic, respond R_2 before S_2 is presented, and so on throughout the series. Further, when these responses precede their former visual stimuli, they are reinforced by the appearance of the visual stimuli. When this becomes possible throughout one perfect anticipatory performance we shall

arbitrarily say that the subject has barely learned the series. This situation is represented in Fig. 2.2

Now, for the sake of a simplified diagrammatic representation, let us use but one component of each stimulus complex. S_1 has

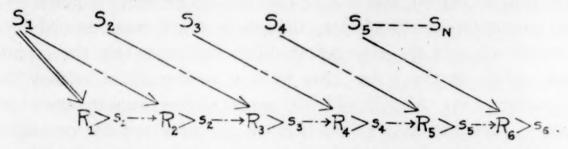


Fig. 2. Visual stimuli S₁, etc., with respective auditory-kinaesthetic stimuli s₁, etc., constitute stimulus complexes or patterns serving to evoke vocal responses R₂, etc.

acquired the ability to evoke R_2 ; we might quite as well use s_1 —-> R_2 ; S_2 has acquired the ability to evoke R_3 , etc.³ This situation is represented in Fig. 3.

So far we have considered only the establishment of immediate excitatory tendencies. We have still to consider the establishment of remote excitatory tendencies. Referring to Fig. 3, Ebbinghaus (6) has demonstrated that S₁ tends not only to evoke

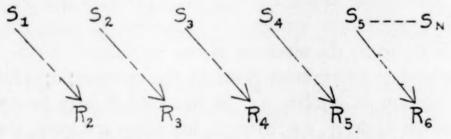


Fig. 3. Conditioned immediate excitatory tendencies.

R₂, but also tends to evoke R₃, R₄, etc., with progressively weaker excitatory tendencies. Likewise, S₂ tends also to evoke R₄, R₅, etc. These remote excitatory tendencies are added to our diagram and appear in Fig. 4, represented by single solid lines.

² This development of the establishment of immediate excitatory tendencies is essentially that of C. L. Hull (9).

 3 S₁ is considered as if presented at the beginning of each trial, so in each trial S₁ \Longrightarrow > R₁ (R₁ being either explicit or implicit) is nothing more than an old language habit.

Now we may consider R_3 , R_4 , etc., as conditioned delayed reactions to S_1 ; in other words, conditioned to a trace effect of S_1 . There is no reason to presume a fundamental difference between delayed conditioning and trace conditioning. In each case the reaction is delayed, and in each case the conditioning is due to the perseveration of stimulation, though in the former case it is a perseveration in the external world as well as within the organism, while in the latter case it is a perseveration within the organism only. Pavlov (27, 40) assumed this organic perseveration. To continue, we have here, in our situation, the necessary conditions for the establishment of excitatory and inhibitory tendencies analogous to those of the well known delayed condi-

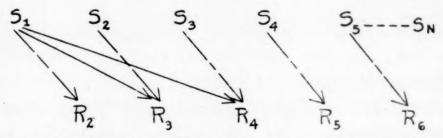


Fig. 4. Conditioned immediate and remote excitatory tendencies.

tioned reaction. When the subject responds R_2 to S_1 the immediately succeeding appearance of S₂ reinforces this conditioned immediate excitatory tendency. However, in case the subject responds R_3 to S_1 the tendency is not reinforced, for S_2 appears. This lack of reinforcement gives us the necessary conditions for the production of inhibition. In this case it is to be considered as inhibition of delay, for, after S2 has come and gone, the remote excitatory tendency S₁——>R₃ becomes appropriate. response R₃ occurring just before S₃ appears is an appropriate response and is reinforced by the appearance of S_3 . In other words, the remote excitatory tendency S_1 —> R_3 perseveres but is held in check by an inhibitory tendency for the appropriate period of delay, after which R₃ is excited as an appropriate delayed reaction to S₁. In such a manner might we treat the increasing degrees of remoteness. Thus, as the learning of the series progresses, remote excitatory tendencies, held in check by inhibitory tendencies for appropriate periods of delay, are established.

These two types of tendency are analogous to the two phases of the delayed reaction as demonstrated by Pavlov (27, 88–105). To repeat, the establishment of the inhibitory tendencies or phases is accomplished by a lack of reinforcement occasioned by the appearance of the intervening stimulus or stimuli. The establishment of the excitatory tendencies, or phases, is accomplished by reinforcement after a period of delay or, in other words, after the intervening stimulus or stimuli have come and gone.

Now, considering the nature of a barely learned series, according to these assumptions, there are established: (1) immediate excitatory tendencies having the nature of higher order, simultaneous or near simultaneous conditioned reactions, and (2) remote excitatory tendencies, held in check for appropriate periods of delay by inhibitory tendencies, thus having the dual nature of the

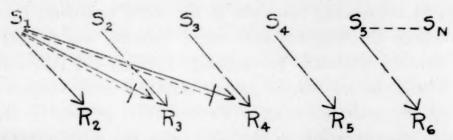


Fig. 5. Conditioned immediate and remote excitatory tendencies and conditioned inhibitory tendencies in the form of inhibition of delay.

well known delayed conditioned reaction. This 'internal' nature of such a barely learned series of acts is represented in Fig. 5.

Such is the major hypothesis. With the hypothesis formulated in this precise manner certain implications are immediately apparent, and certain deductions are made possible. As will be shown in later chapters, this simple hypothesis serves to relate certain phenomena of the conditioned reaction to certain phenomena of serial learning. Its greatest service is in the setting of new, experimentally accessible problems. It serves well as an instrument of controlled and directed, rather than blindly exploratory, research. The chapters immediately following are submitted as exemplifying exploration of this guided character.

CHAPTER II

REMOTE EXCITATION AS A FUNCTION OF THE LENGTH OF THE FORGETTING INTERVAL

I. DEDUCTIONS

Having considered the learning of serial acts in the light of the foregoing hypothesis, let us now turn to a consideration of the probable behavior of these various tendencies beginning with the cessation of practice.4 First, if we were to test for remote excitatory tendencies immediately or soon after the original learning, by relearning test lists at the same exposure interval as that at which the original lists were learned, we should not be able to demonstrate their functioning, because the inhibited delay phases would be of inappropriate lengths with respect to the relearning exposure intervals. Presumably, as will be discussed later, this demonstration would wait upon the shortening of these delay phases with a lapse of time. To explain: if the original lists are learned in a 1-2-3-4-5-6-7-8-9-10-11-12 order, and the derived test lists in a 1-3-5-7-9-11-2-4-6-8-10-12 order. and both are learned using the same exposure rhythm, then, for example, the 1-3 interval in relearning the test list is just one-half the length of the 1-3 interval in the learning of the original series. This, then, is the first deduction. Immediately and shortly following the bare learning or slight overlearning of a series of acts one should not be able to demonstrate the functioning of remote excitatory tendencies. Second, following a relatively longer interval of forgetting one should be able to demonstrate the functioning of remote excitatory tendencies because one would expect a progressive shortening of the periods of delay; or, in more precise terms, one would expect the creeping forward of the

⁴ This monograph deals throughout with remote excitatory tendencies of the first degree. That is to say, the test series are considered as being formulated by the skipping of one stimulus unit.

delayed reactions which are held in check by inhibition of delay. It may well be that this may take the form of the creeping tendency described by Hull (10) and given, by him, much significance, especially in the case of defense reactions. Switzer (32) has recently demonstrated this tendency with the GSR.5 To continue, when this progressive shortening of the period of delay approaches and attains to the point at which the length of delay is appropriate with respect to the relearning exposure intervals. of the test lists, one should be able to demonstrate the functioning of remote excitatory tendencies. This, then, is the second deduction: After a relatively longer interval of forgetting one should be able to demonstrate the functioning of remote excitatory tend-Third, considering now the concurrent weakening of all tendencies, after a still longer period of forgetting, a test of remote excitatory tendencies should reveal their weakening. There appears to be no reason why these remote tendencies should not suffer the various obliterating influences to which immediate excitatory tendencies are subject. This, then, is the third deduction: Following still longer intervals of forgetting one should be able to demonstrate the weakening or possibly the disappearance of remote excitatory tendencies.

Here, then, are three clear-cut deductions based directly and logically upon the foregoing hypothesis.

II. THE EXPERIMENT

(Experiment I)

a. The objective. The major objective of this experiment is to test for the three deductions listed above; in general terms, to study remote excitation as a function of the length of the forgetting interval.

b. The apparatus. The multiple exposure machine used in this and in the subsequent experiments is of the author's design and construction. The machine provides for the accurately timed, successive exposure of serial, nonsense syllable, learning material.

⁵ It should be noted that the immediate excitatory tendencies also have short delays and, under certain conditions, might likewise exhibit a creeping tendency.

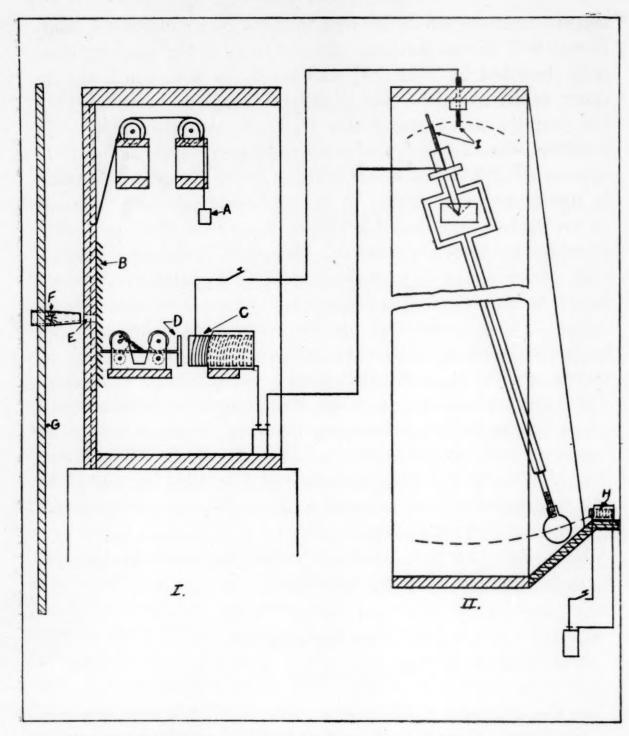


Fig. 6. Multiple exposure apparatus. (I. represents the exposure apparatus. II. represents the timing mechanism. A is a counterweight for the vertically sliding carriage, B. The carriage holds the syllable list. C is an electromagnet which activates the pawl, D, which in turn engages the steel rack on the back of the carriage. E is the exposure window. F is the exposure window light. G is a partition which separates the subject from the apparatus and the experimenter. The timing mechanism, II., is a two second pendulum which regulates the flow of current through the electromagnet, C, by means of the contacts, I. The lower contact is insulated on one side so that the mechanism makes and breaks the circuit once in the course of a double excursion. H is an electromagnetic release for the pendulum. The syllables are exposed as the carriage falls one space every two seconds.

Each syllable is exposed for two seconds. The machine with its significant adjuncts is represented in Fig. 6.

- c. The learning material. The original lists of nonsense syllables from which the test and control lists were derived, were made up by choosing twelve syllables at random from Glaze's (7) sixteen tables. That is to say, each list contained one syllable from each of twelve of Glaze's sixteen tables. This practice was resorted to because the large number of syllables required exceeded the number in any one of Glaze's tables. Otherwise, lists more nearly equated in terms of difficulty might have been obtained by selecting from one table. This random choice was departed from only when the following rules were, by chance, violated. In such cases, a second random choice was made from the same table.
- 1. Each vowel, including y, appeared twice and only twice in any one list.
- 2. The immediate sequence of syllables containing the same vowel was avoided.
 - 3. No initial consonant appeared more than once in a list.
 - 4. No final consonant appeared more than once in a list.
- 5. Sequences wherein the final consonant of one syllable was identical with the initial consonant of the following syllable were avoided.
- 6. Obviously meaningful syllable sequences were excluded. In this manner, twenty-four original lists were formulated. From each of twelve of these lists was derived a remote excitation test list. The technique of Ebbinghaus (6) was used, skipping one syllable. From each of a second group of twelve lists was derived a control list. This was accomplished by the chance permutation of the syllables. Chance permutation was departed from only when original list sequences and first order remote association sequences of syllables appeared. In such cases a second derivation was made. It should be noted here that these derived control lists contain all sorts of forward and backward associations except immediate associations and first order remote associations. They are not control lists in the sense that they contain no associations but only in the sense that they wholly lack

the first order, remote associations which are to be studied and which are prominent in the test lists. The constant error, which might arise from the use of such controls, is at least partially ruled out by the procedure to be described. All lists were lettered upon white cardboard strips in one-fourth inch, black India ink letters. These strips were of such width as to fit snugly into the carriage of the multiple exposure machine.

d. The subjects. The experimental group consisted of ten, unselected, undergraduate students, all of whom were completely naïve with respect to the experimental procedure and its implications.

e. General procedure. 1. All learning and relearning was by the anticipation method.

2. The criterion of mastery was held constant at one perfect anticipatory performance.

3. All comparisons of learning with relearning were made in terms of saving scores. The criterial trials were included in these comparisons.

4. The following instructions were twice read to each subject on the occasion of his first experimental performance:

The subject was seated before the lighted exposure window and the directions were read. "Do you see this little window here? You are to sit here and watch it. I am going to sit on the other side and run the machine. When I start the machine, meaningless three-letter syllables will begin to appear in the window. The first time through you are to read them aloud as they appear. When they have all come and gone, I will reset the machine and start them over again, but this time you are not to read them as they come. Instead, after the first one appears, you are to try to say the second one before it appears. Even if you are not sure, say what you think it is. If you can't say it, just wait for it to come and then, when it does come, try to say the third one before it appears, and so on throughout the list. We shall do this over and over again until you are able to go all the way through the list saying each syllable just before it comes. or in other words, when you see the syllable that comes just before it. Do not practice the syllables while the machine is being reset. Ready?" Then the machine was started and the procedure gone through as indicated. Each time, the subject was given the "Ready?" signal two seconds before the first syllable was exposed. The exposure window light was always extinguished while the machine was being reset, and lighted again just before the "Ready?" signal. A period of eight seconds elapsed between the last exposure of one trial and the first exposure of the next. In no case was it necessary to repeat further or to modify these instructions. As the subject was dismissed at the close of the first learning period these additional instructions were given: "There is one more thing that is very important. Never practice the syllables after you have left the experimental situation."

- 5. The experimenter kept a complete trial-by-trial record of all responses.
- 6. Due to the length of some of the inter-performance periods no attempt was made to control the activity of the subject between learning and relearning performances.
- 7. The time of day at which performances were scheduled was allowed to vary conforming to the convenience of the subjects. The laboriousness of the technique precluded the possibility of controlling this factor.
- 8. Each subject learned two practice lists before beginning the true experimental program.
- 9. In no instance did any subject learn more than one original list and one derived list in twenty-four hours. Thus, between learning and relearning there was no interpolation of similar activities.
- f. Specific procedure. With the practice of the learners, as a group, held constant by rotating the order of the forgetting intervals among the subjects and with possible differences in the syllable lists held constant by rotating the lists among the forgetting intervals, each subject was twice tested for remote excitation in his performance at the end of each forgetting interval. These forgetting intervals were ten minutes, thirty minutes, one hour,

three hours, six hours, and twenty-four hours. Below is the detailed program for each subject, for one forgetting interval:

First day (a) Learn an original list.

(b) Relearn the list in test form.

Second day: (a) Learn an original list.

(b) Relearn the list in control form.

Third day: (a) Learn an original list.

(b) Relearn the list in test form.

Fourth day: (a) Learn an original list.

(b) Relearn the list in control form.

This program was constant for all subjects and for all forgetting intervals except the twenty-four hour interval, where, of course, the relearning of the derived lists would take place on the days following the original learning.

The attempt to control practice effects and possible differences in the learning materials was made according to the scheme outlined below. A set of material consists of the four original lists and the derived lists used for measurement at one interval. Subject I was first tested at the ten minute interval with Set of Material No. 1. Subject II was tested first at the thirty minute interval with Set of Material No. 1. Subject VI was tested last at the six hour interval with Set of Material No. 6. As originally planned, there were twelve subjects in two groups of six. However, two subjects failed to complete the experimental program. To this extent the experiment falls short of the ideal control of practice effects and possible differences in the learning materials.

Subject Sequence

- I 10 mins. (1), 30 mins. (2), 1 hr. (3), 3 hrs. (4), 6 hrs. (5), 24 hrs. (6).
- II 30 mins. (1), 1 hr. (2), 3 hrs. (3), 6 hrs. (4), 24 hrs. (5), 10 mins. (6).
- III 1 hr. (1), 3 hrs. (2), 6 hrs. (3), 24 hrs. (4), 10 mins. (5), 30 mins. (6).
- IV 3 hrs. (1), 6 hrs. (2), 24 hrs. (3), 10 mins. (4), 30 mins. (5), 1 hr. (6).
 V 6 hrs. (1), 24 hrs. (2), 10 mins. (3), 30 mins. (4), 1 hr. (5), 3 hrs. (6).
- VI 24 hrs. (1), 10 mins. (2), 30 mins. (3), 1 hr. (4), 3 hrs. (5), 6 hrs. (6).

III. EXPLANATION OF TABLES AND FIGURES 6

The data summarily presented in Table I were gathered according to the procedure outlined above. From these data the graphs in Fig. 7 were plotted. Each point on these graphs represents the mean of twenty measures. Each point represents the mean relearning advantage of test lists over control lists at the end of the particular forgetting interval. Measurement was accomplished in terms of saving scores. Consider the graph based upon

TABLE I 7 REMOTE EXCITATION AS A FUNCTION OF THE LENGTH OF THE FORGETTING INTERVAL. N=10

		Mean Per cent Saved Upon Relearning				Mean Diff.		
		Test Series		Control Series		(Test minus Control)		
Forgetting Interval	Type of Score	Mean Per cent	Standard Error of the Mean	Mean Per cent	Standard Error of the Mean	Mean Diff.	Standard Error of the Mean Diff.	Critical Ratio
10 mins.	Trials Errors	-0.5 1.3	5.94 5.45	6.3 10.3	9.18 8.87	-6.8 -9.0	11.59 9.25	0.59 0.97
30 mins.	Trials Errors	-7.6 -8.1		-38.3 -54.2	14.91 15.32	30.7 46.1	16.05 15.26	1.91 3.02
1 hr.	{ Trials { Errors	-8.8 -15.2		—12.8 —19.6	5.48 11.92	4.0 4.4	14.52 21.13	0.28 0.21
3 hrs.	{ Trials { Errors	-11.1 -17.0	4.98 6.32	—7.3 —4.4	6.06 6.82	-3.8 -12.6	8.89 10.35	0.43 1.22
6 hrs.	Trials Errors	—7.2 —8.4	9.05 9.02	-3.0 -9.4	6.93 8.08	-4.2 1.0	9.03 10.12	0.47 0.10
24 hrs.	Trials Errors	-4.8 -4.0	10.08 13.98	-0.2 3.2	8.81 7.09	-4.6 -7.2	11.05 17.16	0.42 0.42

error score measurements. The two graphs are in essential agreement. Therefore, we shall consider only one of them in detail. This graph, as a whole, may be considered as representing the functional strength of remote excitation as a function of the length of the forgetting interval.

IV. RESULTS AND INTERPRETATIONS

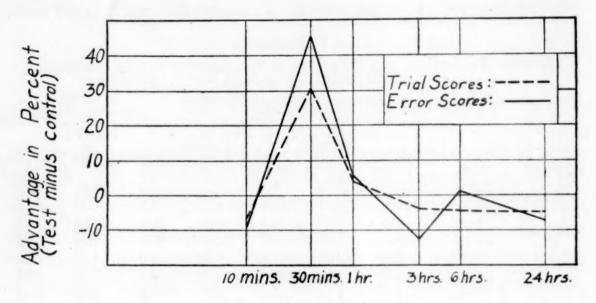
Referring to this graph and to the values in Table I from which it was plotted, note:

1. At the end of the ten minute interval there is a small

⁷ The negative values in the Mean Diff. Column indicate obtained relearning advantages in favor of control lists.

⁶ The summary tables appearing in this article were constructed from complete tables contained in the Appendix of a thesis which is on file in the Library of The Pennsylvania State College.

obtained advantage in favor of the control lists. The value is —9.0. Using the standard error of the difference technique and taking into account the correlation element, the critical ratio is but 0.97.8 Thus, the first deduction is confirmed. Remote excitation is not demonstrated to be functional after a relatively short interval of forgetting. Interpreted in the light of the hypothesis upon which this deduction was based, this result is



Forgetting Intervals

Fig. 7. Remote excitation as a function of the length of the forgetting interval. The negative values of this graph represent obtained relearning advantages in favor of control lists. The abscissa scale in this graph and in the similar graphs to follow is logarithmic in nature.

construed as indicating that the progressive shortening of the inhibited delay phases of the first order remote excitatory tendencies has not attained to such a point that they are appropriate with respect to the relearning exposure rhythm of the test lists.

2. At the end of the thirty minute interval there is a 46.1 point advantage in favor of the test lists. Using the same statistical

⁸ The statistical technique used does not involve the computation of correlation coefficients. Mean saving scores for each subject in test lists and control lists were computed. The mean, control-list, saving score for each subject was then subtracted algebraically from his mean, test-list, saving score. The Standard Error of the Mean Difference, then, is simply the Standard Error of the Mean of the Series of differences so obtained. This technique is used throughout the study in all cases wherein the Means of possibly correlated series are compared. In all other cases involving the Comparison of Means, the conventional formula, $\sigma_{\text{Diff.}} = \sqrt{\sigma^2 M_1 + \sigma^2 M_2}$ is used.

technique, the critical ratio is found to be 3.02. Thus, there is a reliable confirmation of the second deduction. Remote excitation is demonstrated to be functional after a relatively longer interval of forgetting. Interpreted in the light of the hypothesis, the thirty minute interval seems to be of sufficient length to allow the progressive shortening of the inhibited delay phases of the remote excitatory tendencies to a point whereat the latent periods are appropriate with respect to the relearning rhythm.

3. From this thirty minute point on, the advantage of test lists over control lists fluctuates between positive and negative values, but at no point investigated does there occur a reliable difference. Necessarily abstaining from a detailed interpretation of this portion of the graph, the third deduction is confirmed. Remote excitation is not demonstrated to be functional after a still longer interval of forgetting. This may be interpreted as resulting from the weakening or obliviscence of the remote excitatory tendencies. It should be noted here that failure to demonstrate does not insure the total absence of function. However, the point of maximum function is rather clearly indicated to be at the end of the thirty minute interval.

Obviously the foregoing interpretations have been cast intentionally and somewhat gratuitously in terms of the hypothesis under investigation. They remain in need of qualification. For the most part, this qualifying interpretation is left to a later chapter. However, at this point we shall consider these data in comparison with the data from previous studies of remote excitation. First among these studies should be mentioned that of Ebbinghaus (6). Ebbinghaus used a constant learning and relearning rhythm. He tested for remote excitation after a relatively long interval of forgetting, after twenty-four hours, with There is no evidence for remote excitation at positive results. the end of the twenty-four hour interval in the present data. There is no evidence for remote excitation at any point tested beyond the thirty minute point. This apparent abrupt disappearance of remote excitation is probably misleading. As before stated, the failure to demonstrate does not prove the absence of remote excitation. It may well be that the technique used is not sufficiently sensitive. The most remarkable discrepancy between the present data and the data of Ebbinghaus consists of the lack of substantial saving in the relearning of test lists at all points. With comparable derived lists, Ebbinghaus reports a mean saving of 10.8 per cent at the end of twenty-four hours. We should have no evidence for remote excitation at all if it were not for the relatively greater interference in the control lists at the thirty minute point. The author is at a loss to explain this discrepancy. It is necessary to conclude that the results of the present study are not in harmony with the results of Ebbinghaus.

A recent study by Hall (8) offers still greater difficulties. Miss Hall, in an attempt to reconcile the results of Ebbinghaus and Cason (3), so planned her experiment that the latent period for first order remote excitatory tendencies in her test lists remained exactly the same as for the same tendencies in the original lists. Instead of simply skipping one syllable and thereby shortening the period by one-half, as did Ebbinghaus and the present author, she interpolated syllables from other lists, thus maintaining the original latent periods. According to our hypothesis, such a technique should permit the demonstration of remote excitatory tendencies immediately following the original learning. Somewhat contrary to this, she found but slight evidence for remote excitation when she tested immediately. Using the same technique and testing for remote excitation after a forgetting period of one week, she found reliable evidence for remote This latter finding is harmonious with the results excitation. of Ebbinghaus but thus, again, inharmonious with the results of the present study. Here again we find the fundamental discrepancy noted before. Hall's results show a substantial saving in the relearning of test lists, which the results of the present study do not. The saving scores for Hall's subjects averaged 32.2 per cent for the immediate relearning of test lists and 27.1 per cent for the relearning of test lists after one week. Apparently, the potent factors at work in the present study are factors of interference of some sort and the only evidence of remote excitation we have results from a lesser degree of interference in the test lists at the end of the thirty minute forgetting interval.

The graph in Fig. 7 might quite as well be labeled in terms of relative interference or relative associative inhibition.

V. SUMMARY

The data here reported may be interpreted in terms of the hypothesis under consideration. First, remote excitation is not demonstrated to be functional after a relatively brief interval of forgetting. Second, remote excitation is demonstrated to be functional after a relatively longer interval of forgetting. Third, remote excitation is not demonstrated to be functional after a still longer interval of forgetting. In general, with the criterion of mastery held constant, remote excitation, as measured, appears to be a function of the length of the forgetting interval. This fluctuation in measured function may be interpreted as resulting from the decreasing latency of and the obliviscence of remote excitatory tendencies having the nature of delayed conditioned reactions.

Necessary qualifications of the above interpretation are apparent and the author is by no means assured that the effective factors have been identified. Further, the results of this study are considerably out of harmony with the results of previous studies.

CHAPTER III

REMOTE EXCITATION AS A FUNCTION OF THE DEGREE OF LEARNING

I. DEDUCTIONS

There is an obvious corollary to the general conclusion of Chapter II. If it is true that the facilitative effect of remote excitatory tendencies is a function of the interval between the learning of the original and derived lists, when the derived lists involve a shortening of the delay in the S-R sequence, then, assuming that the rate of the shortening of the delay in the remote excitatory tendencies is a function of their fixity at the conclusion of the original learning, the facilitative effect of remote excitatory tendencies in our particular experimental situation should be a function of the degree of learning of the original lists.

To elaborate, let us begin again with the assumptions of the major hypothesis. In a learned series of acts it is assumed: first, that there are established immediate excitatory tendencies having the nature of higher order, simultaneous conditioned reactions; and second, that there are established remote forward excitatory tendencies having the nature of higher order trace or delayed conditioned reactions. The details of this establishment and of the 'inner nature' of such a series will not be repeated A third assumption is to be made here, that the strength or stability of all tendencies is increased by continued learning or overlearning. This assumption is not unfounded. With respect to immediate associations and to simultaneous or near simultaneous conditioned reactions, it is a commonplace. With respect to trace or delayed conditioned reactions, both Pavlov (27, 89) and Switzer (32) have shown that the period of delay, thus the inhibitory tendency, is stabilized by continued conditioning; and also that the reaction, thus the excitatory tendency, is by such means more thoroughly established. Hence, if our second assumption be sound, it logically follows that the progressive shortening of the inhibited phases of remote excitatory tendencies should be retarded by the overlearning of serial acts. other words, it follows that the progressive shortening of the latent periods of remote excitatory tendencies should be delayed by greater degrees of learning. To express this line of thought in terms of Experiment I, assuming that the observed maximum facilitative effect at thirty minutes represents the true maximum, if the original lists had been more thoroughly learned we should have observed a decrease in the measured function of remote excitatory tendencies at the end of the thirty minute forgetting interval. Because, according to these assumptions, we would have retarded the decrease in the latent periods of the remote excitatory tendencies, so that at thirty minutes they would have been still inappropriate with respect to the relearning rhythm of the test lists. Under such conditions the point of maximum measured function should occur later in the course of forgetting, at the end of a longer forgetting interval. This, then, is the deduction which the experiment submitted in this chapter was designed to test: If the observed maximum function at thirty minutes is the true maximum, then, holding the forgetting interval constant at thirty minutes and varying the degree of learning we should be able to show that the greater degree of learning results in a decrease in the measurable function of remote excitatory tendencies.

II. THE EXPERIMENT

(Experiment II)

a. The objective. The objective of this experiment is to test for the deduction expressed above; in general terms, to study remote excitation as a function of the degree of learning.

b. The apparatus. The same apparatus was used as in the preceding experiment.

c. The learning material. The original and derived lists used in this experiment were taken from among those used in the pre-

ceding experiment. In all, there were sixteen original lists, eight of these derived in test form and eight derived in control form.

d. The subjects. This experimental group consisted of twenty unselected undergraduate students, all of whom were completely naïve with respect to the experimental procedure and its implications.

e. General procedure. 1. All learning and relearning was by

the anticipation method.

2. The forgetting interval between the learning of original lists and the relearning of the lists in test or control form was held constant at thirty minutes.

- 3. The degree of learning was varied by requiring the subjects to learn and relearn one-half of the materials to one perfect anticipatory performance and the other half to six perfect performances. The use of these criteria resulted in mean, original learning, trial scores of 9.58 repetitions and 16.59 repetitions, respectively.
- 4. All comparisons of learning with relearning were made in terms of saving scores. In these comparisons only the first criterial trials were included.
- 5. The same instructions were given to this experimental group as were given to the group in the preceding experiment except that the subjects of this group were warned that sometimes they would have to achieve but one perfect anticipatory performance, while at other times they would have to achieve six perfect performances.
- 6. Again the subjects were warned not to practice the syllables after they had left the experimental situation. No other attempt was made to control the activity of the subject between learning and relearning performances.
- 7. The time of day at which performances were scheduled was allowed to vary according to the convenience of the subject.
- 8. Each subject learned two practice lists before beginning the true experimental program.
- 9. In no instance did any subject learn more than one original list and one derived list in twenty-four hours.
- f. Specific procedure. 1. With the interval of forgetting held constant, each subject was tested eight times for remote excita-

tion in his performance at the end of a thirty minute forgetting interval. For four of these tests the criterion of mastery was held constant at one perfect anticipatory performance. We shall speak of this as the first degree of learning. For the remaining four of these tests the criterion of mastery was held constant at six perfect anticipatory performances. We shall speak of this as the second degree of learning.

2. In order to control the influence of possible differences in the syllable lists, the learning materials were systematically alternated between use with measurement involving the first degree of learning and use with measurement involving the second degree of learning. Thus, any syllable list was used one-half of the time with first degree learning and the other half of the time with second degree learning. The details of this alternation are presented later.

In order to control the possible influence of practice effects, four different experimental programs were followed. The first day of each of these programs is outlined below.

Program (a)

Day's program (1)

Learn an original list to the first degree.

After a thirty minute forgetting interval, relearn the list to the first degree in control form.

Program (b)

Day's program (2)

Learn an original list to the first degree.

After a thirty minute forgetting interval, relearn the list to the first degree in test form.

Program (c)

Day's program (3)

Learn an original list to the second degree.

After a thirty minute forgetting interval, relearn the list to the second degree in control form.9

⁹ The second degree relearning was carried to six perfect performances in order that the instructions might be kept constant.

Program (d)

Day's program (4)

Learn an original list to the second degree.

After a thirty minute forgetting interval, relearn the list to the second degree in test form.

Following the first day, there was a systematic alternation of test and control measurements and of first and second degree measurements in each of the four programs. There were twenty subjects in all; five subjects followed each of the four programs. Thus, five subjects began Program (a) with Day's program (1), which was followed by Days' programs (2), (3), and (4), in that sequence. Five subjects began Program (b) with Day's program (2), which was followed by Days' programs (3), (4), and (1). Five subjects began Program (c) with Day's program (3), which was followed by Days' programs (4), (1), and (2). The remaining five subjects began Program (d) with Day's program (4), which was followed by Days' programs (1), (2), and (3). Each subject repeated his program cycle four times.

In order to control possible differences in the materials, the sequence of materials was held constant. To explain, test list No. I, with its original, was always used first in all programs. Thus, it was used one-half of the time in first degree measurements and the other half in second degree measurements. Likewise, control list No. I was always used first in all programs. Thus, it was used one-half of the time in first degree measurements and one-half of the time in second degree measurements.

The other test lists from No. II to No. VIII followed in invariable sequence. The same was true for the eight control lists. Therefore as before stated, each list, with its original was used one-half of the time with the one degree of learning and one-half of the time with the other degree of learning.

III. EXPLANATION OF TABLES AND FIGURES

The data summarily presented in Table II were gathered according to the procedure outlined above.

TABLE II 10

Remote Excitation as a Function of the Degree of Learning, with the Forgetting Interval Held Constant at Thirty Minutes. N=20

Degree of Learning	Type of Score	Type of Series	Mean Per cent Saved upon Relearning		Mean Diff. (Test Minus Control)		Critical Ratio
First	Trials	{ Test { Control	-4.4 -16.7	3.80 6.24	12.3	6.65	1.85
riist	Errors	{ Test { Control { Test { Control	-3.8 -28.3	4.82 7.25	24.5	7.20	3.40
Second	Trials	{ Test } Control	$-3.8 \\ -3.5$	5.10 5.16	-0.3	6.48	0.05
Second <	Errors	{ Test { Control { Test { Control	-9.3 -6.8	6.10 5.93	-2.5	7.04	0.36

Based upon the data in Table II, the column graph designated as Fig. 8 was constructed. The height value of each column represents the mean of eighty measures. Each value represents the mean relearning advantage of test lists over control lists for the degree of learning indicated. Measurement was accomplished in terms of saving scores. As before stated, only the first criterial trial was included in the computation of saving scores.

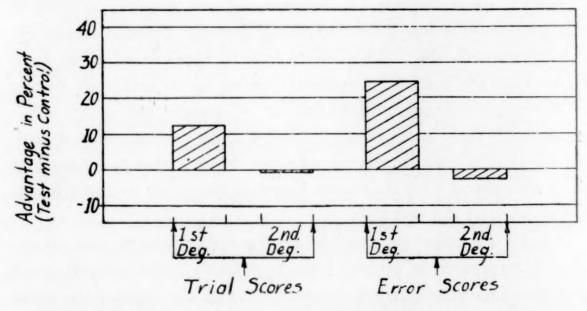


Fig. 8. Remote excitation as a function of the degree of learning. Negative difference values indicate obtained relearning advantages in favor of control lists.

¹⁰ Negative difference values indicate obtained relearning advantages infavor of control lists.

Program (d)

Day's program (4)

Learn an original list to the second degree.

After a thirty minute forgetting interval, relearn the list to the second degree in test form.

Following the first day, there was a systematic alternation of test and control measurements and of first and second degree measurements in each of the four programs. There were twenty subjects in all; five subjects followed each of the four programs. Thus, five subjects began Program (a) with Day's program (1), which was followed by Days' programs (2), (3), and (4), in that sequence. Five subjects began Program (b) with Day's program (2), which was followed by Days' programs (3), (4), and (1). Five subjects began Program (c) with Day's program (3), which was followed by Days' programs (4), (1), and (2). The remaining five subjects began Program (d) with Day's program (4), which was followed by Days' programs (1), (2), and (3). Each subject repeated his program cycle four times.

In order to control possible differences in the materials, the sequence of materials was held constant. To explain, test list No. I, with its original, was always used first in all programs. Thus, it was used one-half of the time in first degree measurements and the other half in second degree measurements. Likewise, control list No. I was always used first in all programs. Thus, it was used one-half of the time in first degree measurements and one-half of the time in second degree measurements.

The other test lists from No. II to No. VIII followed in invariable sequence. The same was true for the eight control lists. Therefore as before stated, each list, with its original was used one-half of the time with the one degree of learning and one-half of the time with the other degree of learning.

III. EXPLANATION OF TABLES AND FIGURES

The data summarily presented in Table II were gathered according to the procedure outlined above.

TABLE II 10

Remote Excitation as a Function of the Degree of Learning, with the Forgetting Interval Held Constant at Thirty Minutes. $N\!=\!20$

Degree of Learning	Type of Score	Type of Series	Mean Per cent Saved upon Relearning		Mean Diff. (Test Minus Control)		Critical Ratio
First	Trials	{ Test { Control	-4.4 -16.7	3.80 6.24	12.3	6.65	1.85
First	Errors	{ Test { Control { Test { Control	-3.8 -28.3	4.82 7.25	24.5	7.20	3.40
Sacrad	Trials	{ Test { Control	$-3.8 \\ -3.5$	5.10 5.16	-0.3	6.48	0.05
Second ?	Errors	{ Test { Control { Test { Control	-9.3 -6.8	6.10 5.93	-2.5	7.04	0.36

Based upon the data in Table II, the column graph designated as Fig. 8 was constructed. The height value of each column represents the mean of eighty measures. Each value represents the mean relearning advantage of test lists over control lists for the degree of learning indicated. Measurement was accomplished in terms of saving scores. As before stated, only the first criterial trial was included in the computation of saving scores.

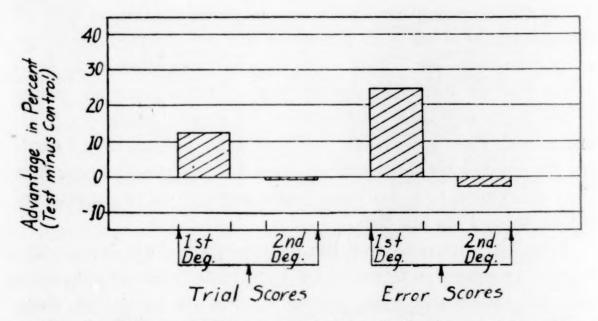


Fig. 8. Remote excitation as a function of the degree of learning. Negative difference values indicate obtained relearning advantages in favor of control lists.

¹⁰ Negative difference values indicate obtained relearning advantages in favor of control lists.

IV. RESULTS AND INTERPRETATIONS

Consider the pair of columns in Fig. 8 based upon error score measurement. The trial score measurements are in essential agreement with the error score measurements. Therefore, we shall consider only the latter in detail. Referring to this graph and to the values in Table II from which it was constructed, note:

- 1. The first degree measurements confirm the second deduction and the second conclusion of Chapter II. With a moderate degree of learning, remote excitation is demonstrated to be functional at the end of a thirty minute forgetting interval. There is a 24.5 point relearning advantage in favor of the test lists. Using the standard error of the difference technique and taking into account the correlation element, the critical ratio of this advantage is 3.40. Thus the confirmation is highly reliable.
- 2. The second degree measurements yield results in confirmation of the deduction which this experiment was designed to test. With a greater degree of learning, the length of the forgetting interval being held constant, there results a decrease in the measurable function of remote excitatory tendencies. a 2.5 point relearning advantage in favor of the control lists. Using the same statistical technique as above, the critical ratio of this advantage is but 0.36, indicating that the obtained advantage is an unreliable one. This result may be interpreted as an indication that the continued overlearning of serial materials results in the more thorough establishment of remote excitatory tendencies with their appropriate inhibited delay phases; thus retarding the progressive shortening of these delay phases and retarding their attainment to a shortened length appropriate to the relearning rhythm of the test lists.

Again, it is obvious that the interpretations have been intentionally expressed in terms of the hypothesis under consideration and that qualifications are necessary. For the most part, these qualifications are left to the following chapter, but here it is pertinent to compare these data with the results of previous studies. Again, the saving scores of the present study are notably inconsistent with the saving scores of Ebbinghaus (6) and of

Hall (8), and the phenomenon of interference or associative inhibition is prominent. The present data offer no evidence of saving in the relearning of either test or control lists. Again, the only advantage of test lists over control lists is in terms of less interference in the relearning of the former.

It is relevant to mention here the results of certain other studies which may be interpreted according to the present hypothesis. It has been suggested by Hall (8) that one of the factors contributing to the negative results of Cason (3) might be the considerable overlearning of the original learning materials. If we might assume that Cason's subjects used a constant relearning rhythm, then, according to our major hypothesis, we might attribute his negative results, in part, to the thorough establishment of the latent periods of the remote excitatory tendencies. This, according to our logic, would prohibit their progressive shortening to a length appropriate to the relearning rhythm of the test materials in the relatively short interval of forgetting allowed. It will be remembered that Cason, using prose materials, tested for remote associations immediately or almost immediately following the cessation of practice in the learning of the original materials.

Ebbinghaus (6) has submitted experimental evidence relevant to the effect of the degree of learning upon the strength of remote associations. Comparing the saving in the relearning of first order derived lists with the saving in the relearning of unchanged lists by expressing the former as a percentage of the latter, he reports that this value decreases as the number of repetitions of the original lists increases. He interprets this as indicating that the continued repetition is relatively more effective in the strengthening of the immediate associations. According to the hypothesis under consideration, we may interpret this result as a decrease in the measurable function of remote excitatory tendencies due to the more thorough establishment of their delay phases, thus to a retardation of the progressive shortening of their latent periods, thus to an increasing inappropriateness of the latent periods with respect to the test relearning rhythm at the time of testing. Ebbinghaus tested after a forgetting interval of

twenty-four hours. In this manner, we may interpret the results of Ebbinghaus.

Lumley (20) has made an interesting contribution to this line of thought. Using a typewriter maze, he has shown that, in the course of learning, anticipatory errors first increase and then, further along in the course of learning, decrease. For example, dividing the whole course of learning into four parts and considering anticipatory errors of all degrees, Lumley reports the following: In the first section of the learning process, 41.6 per cent of the total errors in the section were anticipatory errors. In the second section the value increased to 70.3 per cent. In the third section the value further increased to 77.1 per cent. In the last section the value decreased to 71.9 per cent. These observations have been confirmed in further experiments by Lumley (21, 22) and also by Mitchell (24), whose subjects memorized three-place numbers. Lumley (20) suggests that his results support the conclusion of Ebbinghaus that, as the degree of learning increases, in the course of learning, there are disproportionate effects in the strengthening of immediate and remote associations. In the light of our major hypothesis, the following interpretation may be suggested. If we consider remote excitatory tendencies as a source (not necessarily the only source) of anticipatory errors in learning, the initial increase reported by Lumley and Mitchell may be construed as resulting from the functioning of remote excitatory tendencies in the initial stages of establishment, during which their latent periods, or their inhibited delay phases are relatively unstable. Further, the final decrease in anticipatory errors may be interpreted as indicating the increasing stability of the remote excitatory tendencies with their inhibited delay phases of appropriate lengths.

V. SUMMARY

The results of Experiment II appear to support the results of Experiment I in that remote excitation, as measured by this technique, is again demonstrated to be functional at the end of a thirty minute forgetting interval, the original lists having been learned to a comparable degree of mastery.

The experimental support of the hypothesis here examined is augmented by the confirmation of a fourth deduction. A greater degree of learning results in a lesser mensurability, by this technique, of remote excitation. In accordance with the major hypothesis, this may be interpreted to mean that the overlearning of serial acts results in the stabilization of the remote excitatory tendencies having the nature of delayed conditioned reactions.

If we adhere to this suggested interpretation, remote excitation in serial learning, as measured by this technique, appears to be a function of the degree of learning. In view of the notable discrepancy between the results of this study and the results of previous studies, with respect to the obtained saving scores, this interpretation is not to be urged. Here, as in Experiment I, the dominant factor operating in the relearning of test and control lists appears to be inhibitory in nature and has not been identified.

CHAPTER IV

ASSOCIATIVE INHIBITION

I. INTRODUCTION

To the careful reader an examination of the tables in the two preceding chapters will have revealed a necessity for further interpretations. The reference is to the previously mentioned marked discrepancy between the results of the experiments reported here and the results of Ebbinghaus (6) and Hall (8) as regards saving scores obtained by comparing the learning of original lists with the relearning of the lists in derived form. In order to avoid unnecessary confusion these interpretations have been reserved for the most part for treatment in the present chapter. Due to the fact that the major hypothesis, as stated, does not take into account all of the factors effective in the phenomena to be considered here, we shall depart from the form of the preceding chapters. We shall be concerned with phenomena variously referred to by the terms associative inhibition, habit interference and negative transfer.

II. RELATED STUDIES

The first experimental demonstrations of associative inhibition were contributed by Mueller and Pilzecker (25) and Mueller and Schumann (26). These early investigators formulated the law of associative inhibition somewhat as follows: if x is associated with y, then it is more difficult to associate x with z than it would have been had not the x-y association preceded. These and later studies, involving various types of learning materials, various techniques and both human and sub-human subjects, have demonstrated that associative inhibition is a complex rather than a simple phenomenon. Hunter (15) and, more recently Siipola and Israel (30) have presented summaries of this literature. Of

especial interest to us here are the studies of Bergström (1, 2) and the study made by Kline (17). Using a card sorting technique, Bergström (1) demonstrated that associative inhibitory effects may be negated by practice effects, the resultant being little or no measurable effect. In another experiment, Bergström (2) showed that the resultant associative inhibitory effect decreased as a function of the length of the interval between the first learning and the second learning. If one is guided by the original statement of the law of associative inhibition and considers the factor of obliviscence, this result seems reasonable. That is, if associative inhibitory effects are dependent upon the previous establishment of conflicting habits, the obliviscence of these habits should result in a decrease in the associative inhibitory effects. This has become a commonly accepted principle.

Now let us consider the more recent study made by Kline (17). Using meaningful materials, Kline has demonstrated that the degree of associative inhibition is a function of the degree of learning. Precisely, he has shown: First, that weakly established previous habits have little inhibitory effect upon the establishment of new habits; second, that the inhibitory effect increases with the more thorough establishment of the previous habits; but third, that beyond a certain maximum, the inhibitory effect decreases with the more thorough establishment of the previous habits. This elaborated law of associative inhibition, having been confirmed by other investigators, has become commonly accepted. Insofar as the present author is aware, an obvious corollary to this elaborated law has never been made explicit. this elaboration be sound the generalization of Bergström (2) that the inhibitory effect decreases as a function of the length of the forgetting interval would hold only for slight and intermediate degrees of learning. If this elaboration be sound, then in the third case noted above, wherein the original establishment is very strong, one would expect, in the course of obliviscence of the previously established habits, an initial increase in associative inhibition followed later by a decrease. In other words, when a very strongly established habit which, according to Kline, gives

rise to little associative inhibition, becomes, in the course of obliviscence, a moderately established habit which, according to Kline, gives rise to greater associative inhibition, one would expect an increase in associative inhibition. Further along in the course of obliviscence, when the habit passes below the condition described as moderately established toward the weakly established condition which, again according to Kline, results in little associative inhibition, one would expect a decrease in associative inhibition. This rather striking, deducible phenomenon is apparent in the data discussed below.

This same phenomenon appears to be present in the data of Siipola and Israel (30). At least their data may be so interpreted. These experimenters, using alphabet code materials, have shown that, with the greater degrees of original learning, the interference in the learning of the second task is delayed until relatively later stages in the second learning. In their experiments, the later stages in the second learning took place after considerable forgetting intervals. Thus, the greater interference may be due to factors arising from the obliviscence of the original habit rather than to the stage of learning of the second habit. More likely the obtained resultant effects are causally related to both conditions.

III. EXPLANATION OF TABLES AND FIGURES

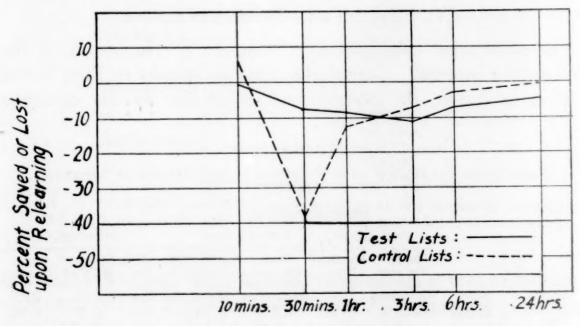
a. Associative inhibition as a function of the length of the forgetting interval. The graphs presented in Figs. 9 and 10 were plotted from the values previously noted in Table I and rearranged in Table III. Fig. 9 was plotted from trial score values and Fig. 10 from error score values. Each point represents the mean per cent saved or lost upon relearning derived test or control lists at the end of the indicated forgetting interval. Each point represents the mean of twenty measurements. Each graph, as a whole, represents resultant values, dependent upon both inhibitory and facilitative factors, varying as a function of the length of the forgetting interval.

TABLE III

Associative Inhibition as a Function of the Length of the Forgetting Interval. N = 10

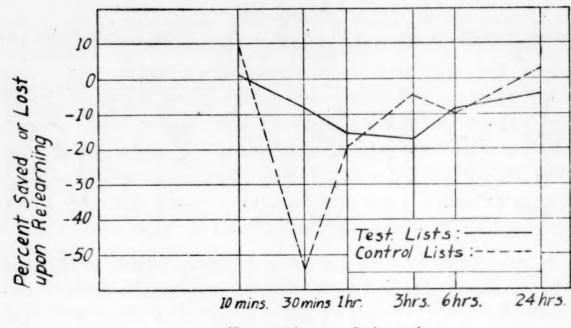
				Forgetting	Intervals		
		10 mins.	30 mins.	1 hr.	3 hrs.	6 hrs.	24 hrs.
Type of Series	Type of Score	Per cent Saved upon Relearning					
Test	{ Trials Errors	-0.5 1.3	-7.6 -8.1	-8.8 -15.2	$-11.1 \\ -17.0$	-7.2 -8.4	-4.8 -4.0
Control	{ Trials Errors	$\frac{6.3}{10.3}$	-38.3 -54.2	-12.8 -19.6	-7.3 -4.4	-3.0 -9.4	-0.2 3.2

b. Associative inhibition as a function of the degree of learning. The column graph in Fig. 11 was constructed from the values previously noted in Table II and rearranged in Table IV. The height value of each column is based upon the mean of eighty measurements. The height value of each column represents the per cent saved or lost upon relearning test or control lists, the period of forgetting being held constant at thirty minutes and the degree of original learning being varied. As before stated, this variation was from learning to one perfect anticipatory performance, in the one case, to learning to six perfect anticipatory



Forgetting Intervals

Fig. 9. Associative inhibition as a function of the length of the forgetting interval (based upon trial score measurements).



Forgetting Intervals

Fig. 10. Associative inhibition as a function of the length of the forgetting interval (based upon error score measurements).

performances in the second case. As a whole, Fig. 11 represents resultant values, presumably dependent upon both inhibitory and facilitative factors, varying as a function of the degree of original learning.

IV. RESULTS AND INTERPRETATIONS

a. Associative inhibition as a function of the length of the forgetting interval. Considering first the graphs in Figs. 9 and 10 as a group, the phenomenon demanded by the corollary

TABLE IV 11

Associative Inhibition as a Function of the Degree of Learning. N = 20

			Degree of	Learning				
		First D	egree .	Second	Degree	C	omparison	
Type of Series	Type of Score	Mean Per cent Saved upon Relearning		Mean Per cent Saved upor Relearning	Error of the	Mean Diff. (1st Deg. minus 2nd Deg.)	Standard Error of the Mean Diff.	Critical Ratio
Test	{ Trials Errors	-4.4 -3.8	3.80 4.82	-3.8 -9.3	5.10 6.10	-0.6 5.5	5.89 7.77	$0.10 \\ 0.71$
Control	{ Trials { Errors	-16.7 -28.3	6.24 7.25	-3.5 -6.8	5.16 5.93	-13.2 -21.5	7.60 8.36	1.74 2.57

¹¹ Negative values in the difference column indicate a decrease in associative inhibition with the increase in the degree of learning.

expressed in section II of this chapter and possibly present in the data of Siipola and Israel (30) is apparent. These graphs consistently exhibit an initial increase in associative inhibition and, later in the course of obliviscence, a decrease. Interpreted in the light of the corollary, it appears that we are dealing with a degree-

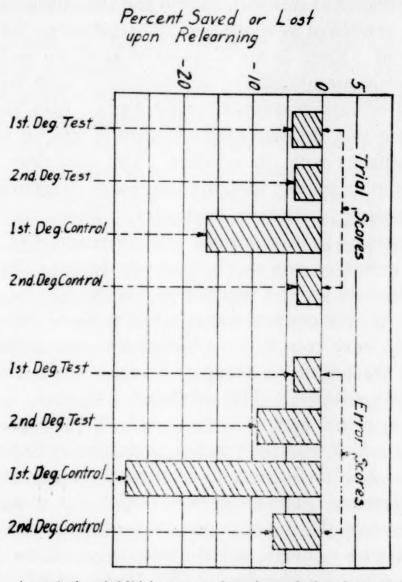


Fig. 11. Associative inhibition as a function of the degree of learning (based upon trial score measurements and upon error score measurements as indicated).

of original learning to be classified as relatively great. Since the measurements by trial scores are in essential agreement with the measurements by error scores let us consider only the latter. Considering now the error score graph representing associative inhibition in the relearning of derived control lists, note the marked increase in this function between ten minutes and thirty

minutes and, following this increase, the consistent decrease. The magnitude of the increase is from a plus 10.3 value to a minus 54.2 value. The magnitude of the decrease from the thirty minute point to the twenty-four hour point is from the minus 54.2 value to a plus 3.2 value. There are doubtless several factors operative in this phenomenon and the values, as measured, are to be considered as resultants. The following list of factors is suggested. Each factor is classified as to its probable influence, either as an inhibitor or as a facilitator, with respect to the relearning of lists in derived control form. Also, the direction of variation as a function of the forgetting interval is indicated.

- 1. Familiarity with the syllables. This facilitator will suffer a decrease in its potency with the progress of forgetting.
- 2. Immediate excitatory tendencies. These inhibitors will suffer a decrease in their potency with the progress of forgetting.
- 3. All orders of remote excitatory tendencies. These inhibitors will likewise suffer a decrease in potency.
- 4. The spontaneous recovery of other types of irrelevant reactions which have been first established and then inhibited in the course of the original learning; for example, the correction of faulty pronunciations. This inhibitory influence, if it exists, might be expected to first increase with the obliviscence of the inhibitions which hold the reaction tendencies in check, and then to decrease with the continued obliviscence of the reaction tendencies themselves. The assumption is that this complex process is analogous to the establishment, the experimental extinction, the spontaneous recovery, and the obliviscence of the conditioned reaction, as described by Pavlov (27, 16–32, 48–67).

In addition, there may be other factors less predictable as to their function. This list is not submitted as an exhaustive one. Hall (8, 73) has suggested that factors 2 and 3 above give rise to some sort of inhibition because of the fact that the old immediate and, in some cases, the remote bonds, having become inappropriate in the derived lists, must be broken. Hull (14) has recently expressed this thought in terms of a "frustration hypothesis". To quote from Hull:

"The term frustration is used here to indicate any situation in which an acquired excitatory tendency . . . is for any reason prevented from evoking its accustomed reaction. The hypothesis is that, under such circumstances, internal inhibitions will be developed which will manifest substantially the same characteristics as result from the experimental extinction of conditioned reactions."

If this hypothesis of Hull's be sound we might do well to eliminate the term associative inhibition except in such cases as the present one wherein it is probable that several factors contribute to the resultant interference effect.

Lack of experimental evidence makes it impossible to give these factors significant relative weights. Therefore, the author confines himself to suggesting that the temporal course of associative inhibition, as indicated by the graph under consideration, is a complex function, the resultant effect of the factors listed. The author wishes to point out that, if factor number 4 is a reality, and a strongly influential one, it might account for the observed fluctuation of associative inhibition from slight to relatively greater, and then to relatively less.

Let us turn now to a consideration of the error score line-graph representing associative inhibition in the relearning of derived test lists. Note the general similarity of this graph to that concerned with control lists. In this graph there is a consistent increase in associative inhibition from a plus 1.3 value at the ten minute point to a minus 17.0 value at the three hour point. Beyond the three hour point, there is a consistent decrease in associative inhibition to a minus 4.0 value at the twenty-four hour point. Note especially the difference between the two graphs at the thirty minute point. Referring back to Table I, note that the only statistically significant difference between test list values and control list values is this difference found at the thirty minute point. Subtracting the control list value from the test list value, the magnitude of the difference was found to be a plus 46.1. The ratio of this difference to its standard error was found to be 3.02. In Chapter II, we have already interpreted this difference as an index of the measurable potency of first order, remote excitatory tendencies. Our interpretation of the test list graph is no different from our interpretation of the control list graph except for this one factor. In the relearning of derived test lists the decreasing latency of first order, remote excitatory tendencies contributes an added facilitating influence which, in the temporal course of forgetting, first increases to a maximum with the obliviscence of the inhibitory delay phases and which, beyond this maximum, decreases with the continued obliviscence of the excitatory phases. The lesser degree of associative inhibition in the relearning of test lists at the thirty minute point, as compared with associative inhibition in the relearning of control lists at the same point, is attributed to this facilitating factor which, according to the major hypothesis, is not present in the control lists.

b. Associative inhibition as a function of the degree of learning. Here we shall be concerned with an interpretation of the values of Table IV and Fig. 11 consistent with the major hypothesis and with our interpretation of the data in the immediately preceding section.

Let us consider first the variation in associative inhibition in the relearning of derived control lists. It is evident that the greater degree of learning (second degree) results in a decrease in associative inhibition. Measured by trial scores, this difference has a value of 13.2. The standard error of this difference is 7.60 and the critical ratio is 1.74. This decrease is a reasonably significant one. The error score measurements are in essential agreement with the trial score measurements, and the decrease is considerably more reliable. The decrease value is 21.5 and the critical ratio between this mean decrease value and its standard error is 2.57. Measured by trial scores, there is little evidence of associative inhibition in the relearning of control lists with the second degree of learning. The obtained value is a minus 3.5. Measured by error scores, there is again slight evidence of associative inhibition under these conditions. Here the obtained value is a minus 6.8. This result appears to support Kline's (17) conclusion that extremely well established habits result in little or no measurable associative inhibition.

Evidently, by raising the learning criterion from one perfect anticipatory performance to six perfect anticipatory performances, we have proceeded from a well established habit to a still more thoroughly established habit. It should be noted here that, according to the corollary demanding an initial increase in associative inhibition with the progress of the obliviscence of strongly established habits, one would here again expect such an increase with a period of forgetting somewhat longer than thirty minutes.

For a detailed interpretation of the experimental outcome noted above, let us refer again to the four factors listed in the immediately preceding section.

1. Familiarity with the syllables. This facilitator will increase in potency with a greater degree of learning.

2. Immediate excitatory tendencies. These inhibitors will increase in potency with a greater degree of overlearning.

3. All orders of remote excitatory tendencies. These inhibitors will likewise increase in potency.

4. The spontaneous recovery of other types of irrelevant reactions. This spontaneous recovery, inhibitory in its influence, will be retarded with a greater degree of learning. The greater degree of learning presumably results in their more thorough extinction. It is even conceivable that this extinction might attain to such a degree that spontaneous recovery would never occur.

Since bases upon which to weight these suggested factors are lacking, the only possible interpretation of the observed decrease in associative inhibition as a function of increased degree of learning is a general one. The increased overlearning of derived control lists accomplishes a readjustment of the relative potencies of facilitating and inhibiting influences such that there results little or no measurable associative inhibition.

Let us now turn to the values in Table IV which represent associative inhibition in the relearning of derived test lists as a function of the degree of learning. Measured by trial scores, there is no significant difference between the value obtained with the first degree of learning and the value obtained with the second degree of learning. There is an obtained decrease value

of minus 0.6, but the ratio of this difference to its standard error is only 0.10. Measured by error scores, there is an obtained increase value of plus 5.5, but the critical ratio here is only 0.71. It is legitimate to conclude that these data show no significant change in the resultant associative inhibition in the relearning of derived test lists with an increase in the degree of learning. accordance with the major hypothesis and with preceding interpretations, this outcome may be interpreted as follows: In the greater overlearning of test lists there is a decrease in the potency of the facilitating influence arising from first order remote excitatory tendencies. This decrease in potency is attributed to the more thorough establishment of the delay phases of these remote excitatory tendencies. This more thorough establishment prohibits their progressive shortening to a length appropriate to the relearning rhythm of the test lists in the limited, thirty minute forgetting interval allowed. Consistent with our interpretation of the control list values, the decrease in the potency of this facilitating factor negates the resultant value which gave us a decrease in associative inhibition with the greater overlearning of control lists. Combining the above interpretations into one expression, the observed discrepancy between (1) the significant reduction in associative inhibition in the relearning of control lists more thoroughly overlearned and (2) no significant reduction in associative inhibition in the relearning of test lists more thoroughly overlearned, may be interpreted as being due to the varying influence of first order, remote excitatory tendencies, having the nature of delayed conditioned reactions.

V. SUMMARY

The data from Experiments I and II have been reconsidered in an attempt to throw light upon the phenomena of associative inhibition.

On the basis of the data from Experiment I there is suggested a new 'law' of associative inhibition. When there is a relatively great overlearning of a series of acts, the associative inhibitory effect of such a series upon the learning of the same or similar acts in a disturbed serial order, may first increase and then decrease as a function of the length of the forgetting interval.

At present it is impossible to predict whether or not this corollary will hold for all types of learning materials and practices. Very likely it will not. However, if we accept the foregoing interpretations, serial nature and the consequent presence of remote excitatory tendencies do not appear to be fundamentally essential conditions.

The data from the relearning of control lists in Experiment II confirm the findings of other experimenters that, in cases of thoroughly established habits, associative inhibition decreases as a function of increasingly thorough establishment.

The conclusions from Chapters II and III are reiterated in the consideration of remote excitation as a factor in associative inhibition. This factor is classified as an associative inhibitor in the relearning of derived control lists. In contrast with this, the influence of first order remote excitations is considered as an associative facilitator in the relearning of derived test lists. It is suggested that other factors are effective in the phenomena of associative inhibition and associative facilitation. These other factors are:

- 1. Familiarity with the syllables, a facilitator.
- 2. Immediate excitatory tendencies, considered as inhibitors.
- 3. The spontaneous recovery of irrelevant reactions which have been first established and later extinguished in the course of learning. This factor, if it exists, is considered as being inhibitory in its influence.

In agreement with other writers, it is concluded that the phenomenon of associative inhibition, as observed and measured, is the resultant effect of several influential factors, rather than a simple function.

Here it is necessary to say that these interpretations, though consistent, are somewhat gratuitous and that there is no satisfactory assurance that the effective factors have been identified.

The above reconsideration of the data from Experiments I and II fails to provide any explanation of the discrepancy between these data and the data of Ebbinghaus (6) and Hall (8) as regards the magnitude of saving scores. The need for further investigation is apparent.

CHAPTER V

SERIAL LEARNING ORDER AS A FUNCTION OF PRACTICE

I. INTRODUCTION

Experiment III, reported in this chapter, was suggested by the casual observation that, as the subjects used in Experiments I and II proceeded through their experimental programs, they appeared to be learning more and more extensively from the anterior end of the series. Or, in terms of serial learning order, the primacy effect appeared to be more pronounced later in the subject's experience with the experimental technique than earlier.

II. THE EXPERIMENT

(Experiment III)

Actually, Experiment III does not represent a new experimental procedure. Certain data taken from Experiments I and II are reconsidered in an attempt to discover whether or not serial learning order varies as a function of the learner's practice.

III. EXPLANATION OF TABLES AND FIGURES

Table V and Fig. 12 represent a summary treatment of data taken from the first twelve original-series, learning records of the thirty subjects used in Experiments I and II. They represent a comparison of the composite learning order for the first six series learned and the learning order for the second six. It should be remembered that each original series learned was learned subsequently in a derived form, so the practice of these learners was approximately twice as extensive as that represented by these twelve original lists. Each serial position point in Fig. 12 is based upon a mean error score computed from one hundred and eighty learning records. Each graph, as a whole, represents the serial

TABLE V

SERIAL LEARNING ORDER AS A FUNCTION OF PRACTICE. COMPOSITE LEARNING ORDER IS EXPRESSED IN TERMS OF THE MEAN NUMBER OF ERRORS AT EACH SERIAL POSITION. N=30 (Adult Subjects)

Comparison

	Ratio		5.21
undard Error f the	Diff.		1.17 5.21
Mean Sta Diff. F	'nd Six)		6.1
Standard Error (Mean 2	1.33	1.48
Mean Per cent of the Total Errors in the Standard Diff. F Anterior Error (1st Six o	the Series	45.5 1.33	39.4 1.48 6.1
1	Total	110.1	7.0 7.0 6.6 4.9 2.9 53.6
	=	4.4	2.9
	10	7.5	4.9
	9 10 11	11.6	9.9
	00	13.9	7.0
tion	7	14.3 13.9 11.6 7.5 4.4	7.0
Serial Posi	9		
Seri	S	3.9 6.6 9.1 11.4 13.5 13.9	1.6 2.4 3.5 4.9 6.3 6.5
	4	11.4	4.9
	3	9.1	3.5
	2	9.9	2.4
	1		
	Series	First six learned	Second six learned

TABLE VI

Serial Learning Order as a Function of Practice. A Comparison Based upon the Percentage of Errors Made in the Anterior Halves of the Series. N=30 (Adult Subjects)

Series Mean Percent of Error in the Anterior Halves of	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th	12th
the Series	48.8	45.5	45.5	46.	9.94	40.5	38.3	41.8	9.04	36.8	41.2	38.0
Means by Groups of Three46.6		9.94			4.5			40.2			38.7	44.5 40.2 38.7

learning order for the first six series learned or the second six series learned, as indicated.

Table VI and Fig. 13 are intended to demonstrate that the changing order of learning, as a function of the practice of the learner, is continuous. In Table VI, the per cent of error in the anterior half of the series is shown for each of the twelve stages of practice. The irregular, solid line in Fig. 13 is plotted from these values. The abscissa scale represents the stages of practice. The smoothed, broken line represents simple means, combining three stages of practice.

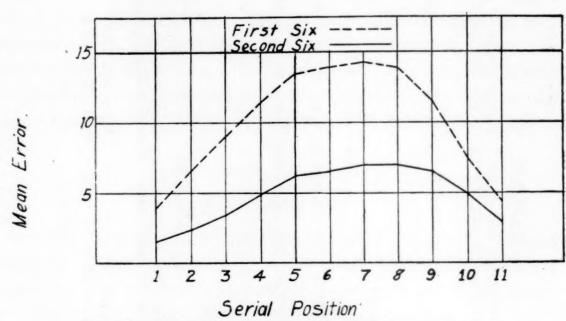


Fig. 12. Serial learning order as a function of practice.

IV. RESULTS AND INTERPRETATIONS

Considering first Table V and Fig. 12, note that the primacy effect is more prominent in the learning of the second six lists than in the learning of the first six. Referring to Table V, which offers a statistical comparison based upon the mean percentage of error in the anterior halves of the series, note that this group of thirty subjects made 45.5 per cent of their errors in the anterior halves of the first six series learned. Compared with this value, note that they made only 39.4 per cent of their errors in the anterior halves of the second six series learned. The critical ratio of this 6.1 per cent difference to its standard error of 1.17 is 5.21. This difference is a highly reliable one, and it seems conclusive that

adult subjects do learn more and more extensively from the anterior end of the series with the extension of practice.

An inspection of Table VI and Fig. 13 reveals that the change in learning order noted in our gross comparison of the first six series learned with the second six learned is a continuous process. The mean per cent of error in the anterior half of the series

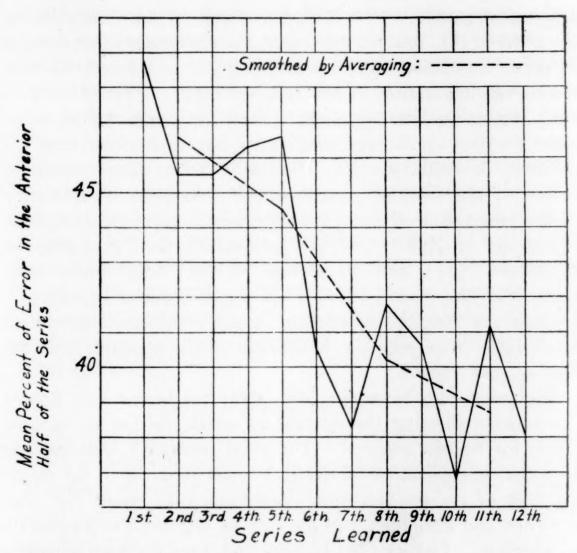


Fig. 13. Demonstrating the continuous nature of the change in learning order as a function of the practice of the learner.

varied from 46.6 in the first quarter of the practice represented to 38.7 in the last quarter or in the learning of the fourth group of three lists.

Several possible interpretations of this phenomenon of changing learning order suggest themselves. It may be that the 'memory span' of the subject increases with practice, thus allowing him to learn the syllables in larger and larger groups. With such an interpretation it would be necessary to assume that this is most effective in learning the anterior half.

Various hypotheses have been put forward in the interpretation of serial learning order and various factors have been shown to be influential. Considering first, maze learning by the rat, Peterson (28) and Dashiell (4) have pointed out the influence of orientation factors. Hull, with his concept of the "habit-family hierarchy"(11), has recently offered a systematic interpretation of these orientation factors. DeCamp (5) and Kuo (18) have shown the importance of the relative length of the blind alley with respect to learning order. Most rat maze studies show some evidence of a regressive order of elimination, thus the influence of serial position. Spence (31) has lately offered a review of this literature and has made a brilliant interpretation of the phenomena of serial learning order, using the systematic hypotheses of Hull (11, 12, 13), particularly the "goal gradient hypothesis" (13), and the concept of the "habit-family hierarchy"(11) mentioned above. The "goal gradient hypothesis" assumes a differential strength of conditioning in a sequence of acts which varies with the remoteness of the segment from the final or goal reaction.

Beginning with Meumann (23), there has been a long line of experiments showing the relation of serial position to learning order for human subjects. The most consistent outcome has been the demonstration of the centripetal progress in the establishment of correct reactions. Meumann (23) noted this phenomenon and attributed it to the varying degree of concentration of attention. Lumley (20) believes that it results from grouping practices of the subjects. Welch and Burnett (33) noted a marked reduction in the primacy effect when they gave their subjects specific instructions not to rehearse the earlier syllables of a series while the later ones were being presented. They obtained an order of learning quite like the regressive order of the rat. Robinson and Brown (29) reasonably doubt the effectiveness of this sort of instruction. Further, they point out that the results of Welch and Burnett may well be a function of the manner in

which the records were taken. In their own experiments they used the anticipation method and required their subjects to spell the syllables, intending to control the attention of the learner. Speaking of the primacy effect, they conclude, "We do not believe that this is due to the rehearsal of the first few syllables during the presentation of the later ones."

Whatever the factors are, there are evidently several of them. The results of the present study demand the existence of a factor which may reasonably vary with the practice of the learner. The author wishes to suggest that this variable factor may be some sort of symbolic supplementation on the part of the subject, probably embracing grouping practices, rehearsal, and verbal associations, both logical and arbitrary. This suggestion needs to be supplemented by two assumptions: first, that this supplementation increases as a function of practice; and second, that this increase is most effective in the learning of the anterior half of the series. It should be noted here that the first assumption is contradictory to an observation reported by Meumann (23). He reports that, with continued practice, subjects make less and less use of this sort of supplementation. The brief protocols taken in the present study reveal nothing significant for the solution of this contradiction.

V. SUMMARY

The data treated in this chapter exhibit a phenomenon of serial learning order which, insofar as the author is aware, has not been reported heretofore. As measured by the technique described, serial learning order varies as a function of the learner's extended practice. With continued practice in the learning of serial, nonsense syllable materials, the learner learns more and more extensively from the anterior end of the series; the primacy effect becomes increasingly prominent. A guess is hazarded that this phenomenon is dependent upon the extent to which the learner employs supplementary, symbolic acts in his learning technique.

CHAPTER VI

SERIAL LEARNING ORDER AS A FUNCTION OF AGE

I. INTRODUCTION

To speak of learning order as a function of age is most ambiguous. However, there seems to be no alternative because the true variable factors effective in the experiment to be reported have not been identified.

In the preceding chapter it has been suggested that the increasing primacy effect resulting from continued practice may depend upon the subject's increasing use of supplementary symbolic acts in his learning technique. The author wishes to suggest here that the difference noted between earlier and later learning order is at least superficially and qualitatively similar to the frequently observed difference between human subjects and lower animals as regards serial learning order. Expressed in terms of the elimination of errors, it has been shown that the rat learns predominantly from the goal end of a series of acts toward the anterior end; and, contrasted with this, it has been demonstrated that the human subject learns characteristically from the two extremes of the series toward the middle. The study made by Husband (16), in which he compared human adults and white rats in maze learning, clearly shows this difference. In spite of a certain amount of diversity in experimental outcomes, this difference has been quite consistent in its appearance. little if any doubt that the human subject makes more extensive use of symbolic supplementation in his learning technique than does the lower animal. The inference, then, is that the observed difference between human and lower animals as regards serial learning order is dependent, at least partially, upon the same factors as that difference between earlier and later learning order for human subjects reported in the preceding chapter.

rather plausible series of conjectures led to the planning of Experiment IV, reported in this chapter.

Leaving out of account the suggested specific factor of symbolic supplementation, one might reason that if serial learning order is a function of phylogenetic status it may be also a function of ontogenetic status. The following experiment was designed to test this conjecture.

II. THE EXPERIMENT

(Experiment IV)

- a. The objective. This experiment was designed to test the conjecture ventured at the close of the preceding section; in general terms, to measure serial learning order as a function of the age or ontogenetic status of human subjects.
- b. The learning materials. The ten-unit nonsense syllable lists used in this experiment were formulated in a manner similar to that employed in the construction of those used in the preceding experiments.
- c. The subjects. With the Kuhlmann-Anderson Group Intelligence Test, ten inferior boys were selected from the seventh grade of a public school. By means of the same test, ten superior boys were selected from the eleventh grade of the same school. This technique was used in an attempt to secure groups as widely separated in ontogenetic status as possible. There were irrelevant reasons for selecting from these two particular grades. The mean mental ages for these groups were twelve years and five months, and nineteen years and two months, respectively.
- d. General procedure. 1. The same apparatus was used as in the preceding experiments.
- 2. The same instructions were given to these subjects as were given to the adult subjects in Experiment I.
- e. Specific procedure. 1. Each subject learned a practice list of five syllables before beginning the true experimental program.
- 2. Each individual in these two groups learned five ten-unit, nonsense syllable series by the anticipation method. A period of two weeks elapsed between the learning of each series and the learning of the following one.

3. In order to control possible differences in the learning materials, two subjects within each group of ten subjects learned the five lists in a 1, 2, 3, 4, 5 sequence; two subjects learned them in a 2, 3, 4, 5, 1 sequence two in a 3, 4, 5, 1, 2 sequence; two in a 4, 5, 1, 2, 3 sequence; and, lastly, two subjects learned the five lists in a 5, 1, 2, 3, 4 sequence.

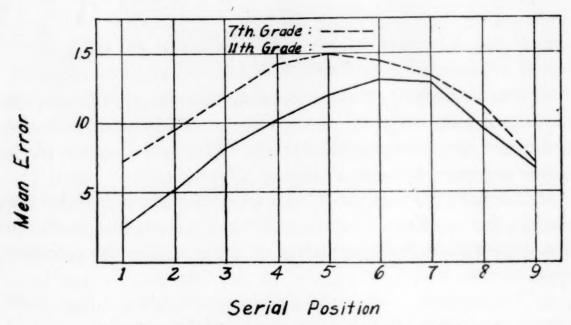


Fig. 14. Serial learning order as a function of age.

4. A complete trial by trial record of all reactions was kept for each learning performance.

III. EXPLANATION OF TABLES AND FIGURES

The data presented in Table VII were obtained according to the above procedure. From these data the graphs appearing in Fig. 14 were plotted. Each point on these graphs represents the mean of fifty measurements. Each point represents the mean error score for the serial position and for the type of subject indicated.

Table VIII represents a reconsideration of the data from Experiment IV. Specifically, this table represents a simplified comparison of the composite learning order for the first two series learned with the learning order for the last two series

TABLE VII

SERIAL LEARNING ORDER AS A FUNCTION OF AGE. COMPOSITE LEARNING ORDER IS EXPRESSED IN TERMS OF THE MEAN NUMBER OF ERRORS AT EACH SERIAL POSITION. N=10 (School Subjects)

S CONDI	110	NE
Critical	Ratio	4.92
Standard Error of	Mean	2.36
Mean Diff. (7th Grade	Grade)	11.6
Standard Error of	Mean 1.60	1.73
Mean Per cent of the Total Errors in the Anterior	the Series	34.8
2	Total 104.2	80.4
	7.2	8.9
	8	9.6
tion		
ial Posi	15.0	12.0
Ser	14.2	10.2
	3,11.9	8.1
	9.5	5.2
	7.3	2.5
Section	Seventh Grade	Eleventh Grade
	Mean Per cent of the Total Standard Errors in Error Serial Position Mean Standard Error Diff. Error the Arror of (7th Grade of Diff. Oritical	Mean Per cent of the Total Standard Error Diff. Error the Anterior of (7th Grade of Critical Half of the minus 11th the Critical He Series Mean Grade) Mean Ratio

learned, the data from the two age groups being considered separately.

IV. RESULTS AND INTERPRETATIONS

Referring to Table VII and to Fig. 14, note that the table and the graph indicate that the seventh grade boys found the fifth unit most difficult, while the eleventh grade group found the sixth unit most difficult. When the learning orders of the two groups were compared by computing the mean percentage of the total errors in the anterior halves of the series learned by each group it was found that the eleventh grade group made 34.8 per cent of their

TABLE VIII

Serial Learning Order as a Function of Practice. A Comparison Based upon the Percentage of the Total Errors Made in the Anterior Half of the Series. N=10 (School Subjects)

Group Series	Mean Per cent of the Total Errors in the Anterior Half of the Series		d Mean Diff. (First Two minus Last Two)	Standard Error of the Mean Diff.	Critical Ratio
Seventh First Two Learned		2.13			
Grade Last Two Learned	39.9	2.98	4.2	3.04	1.38
Eleventh JFirst Two Learned	44.0	2.33			
Grade Last Two Learned	33.6	2.54	10.4	3.04	3.42

errors in the anterior halves of the series, while the seventh grade group made 46.4 per cent of their errors in the anterior halves. The critical ratio between this 11.6 per cent difference and its standard error of 2.36 is found to be 4.92. Thus, there is reliable evidence that serial learning order, as measured by this technique, varies with the age of the learner.

An inspection of Table VIII reveals some confirmation of the results of Experiment III in Chapter V, wherein it was shown that the primacy effect increased as a function of the extended practice of the learner. The seventh grade boys made 44.1 per cent of their errors in the anterior halves of the first two series learned as compared with 39.9 per cent made in the anterior halves of the last two series learned. The ratio of this 4.2 per cent difference to its standard error is rather small, being 1.38. Therefore, this result is confirmatory only in the sense that there

is a difference in the same direction. The eleventh grade boys made 44.0 per cent of their errors in the anterior halves of the first two series learned and 33.6 per cent of their errors in the anterior halves of the last two series. The ratio of this 10.4 per cent difference to its standard error is 3.42. Thus, there is a highly reliable confirmation of the conclusion in Chapter V, which states that the primacy effect increases with the extent of practice of the learner.

V. SUMMARY

The data examined in this chapter offer some justification of the conjecture submitted at the close of the chapter's introduction. Serial learning order appears to be in some way a function of the age or ontogenetic status of the learner. A group of seventh grade subjects exhibits the primacy effect in serial learning order to a lesser degree than does a group of eleventh grade subjects.

Both groups of subjects studied in the present experiment exhibit a changing learning order, characterized by an increase in the primacy effect, as a function of the extent of practice.

This result agrees with the outcome of Experiment III in Chapter V. Again a guess is hazarded that these phenomena are causally related to the extent to which the learner employs supplementary, symbolic acts in his learning technique. Neither here nor in the preceding chapter is there any reliable assurance that the independent variables effective in the phenomena described have been precisely identified. The isolation of these variables waits upon more extensive investigation. However, it remains conclusive that the gross factors of age and practice must be taken into account in the future study of serial learning order.

CHAPTER VII

RECAPITULATION AND CONCLUSION

This writing began with the simple assumption that remote excitatory tendencies have the dual nature of trace conditioned reactions. In other words, it was assumed that remote excitatory tendencies are characterized by (1) an excitatory phase and (2) an inhibitory delay phase. This major assumption suggested a host of feasible tests of its validity and directed the planning of the two major experiments, I and II. The results of these experiments may be interpreted as supporting this assumption. Further, the hypothesis appears to be of some service in the interpretation of certain of the phenomena of serial learning previously observed.

The two minor experiments, III and IV, were suggested by incidental observations made during the progress of the major experiments.

A detailed review would be unnecessarily repetitious. Each preceding chapter includes its own summary reporting both results and interpretations. For the benefit of the cursory reader the experimental outcomes are repeated here, but without interpretations.

- 1. As measured by a modified Ebbinghausian technique, remote excitation appears to be a complex function of the length of the forgetting interval. The function, as measured, exhibits an initial increase followed by a decrease.
- 2. Measured by the same technique, remote excitation appears to be a complex function of the degree of original learning. Overlearning the original materials appears to render remote excitation less measurable, when the interval of forgetting is held constant.
 - 3. There appears a marked discrepancy between the results

referred to in (1) and (2) and the results of previous studies of remote excitation. In neither of these experiments was there any substantial saving in the learning of derived series.

- 4. Associative inhibition in both test and control lists is shown to vary with the length of the forgetting interval. In these experiments, associative inhibition exhibits an initial increase, followed by a decrease in the course of forgetting. The initial increase is most marked in the control lists.
- 5. Associative inhibition in the derived control lists appears to vary inversely with the degree of original learning. There occurred a significant reduction in associative inhibition with the greater degree of original learning. No significant change of this sort was observed in the case of derived test lists.
- 6. Serial learning order is demonstrated to vary with the age of the learner. Older subjects learn more extensively from the anterior end of the series. The primacy effect is less prominent in the learning order of the younger subjects.
- 7. Serial learning order is demonstrated to vary with the extended practice of the learner. The later lists learned exhibit the primacy effect to a greater extent than do those lists learned earlier in the experience of the subject.

These phenomena are all interpreted in the light of the hypotheses and conjectures under consideration. Insofar as these and other phenomena of serial learning may be deduced from the assumptions of the hypotheses, they may be considered as confirmatory.

The author is fully aware of the loose articulations, at certain points, in the logic of this treatment. At the present preliminary stages in the evolution of this line of thought, this falling short of the clear-cut ideal may be attributed to the complexity of the phenomena involved and to certain lacunae in the experimental evidence.

Considering the interpretations submitted in the various chapters, the only assurance that the fundamental, independent variables have been identified is that indirect assurance afforded by some degree of internal consistency among the observations which are related according to the hypotheses. These hypotheses are submitted as working hypotheses, for criticism and for further experimental testing. Whether or not they prove sound, they serve as fruitful sources of new questions, new experiments, possibly of new discoveries.

BIBLIOGRAPHY

- 1. Bergström, J. A. The relation of the interference to practice effect of an association. Amer. J. Psychol., 1894, 6, 433-442.
- 2. Bergström, J. A. Experiments upon physiological memory by means of the interference of associations. *Amer. J. Psychol.*, 1893, 5, 356-369.
- 3. Cason, H. Specific serial learning: a study of remote forward association. J. Exper. Psychol., 1926, 9, 299-324.
- 4. Dashiell, J. F. The need for analytical study of the maze problem. Psychobiol., 1920, 2, 181-186.
- 5. DECAMP, J. E. Relative distance as a factor in the white rat's selection of a path. Psychobiol., 1920, 2, 245-253.
- 6. EBBINGHAUS, H. Memory. (Tr. by Henry A. Ruger and Clare E. Bussenius.) New York: Teachers College, Columbia Univ., 1913.
- 7. GLAZE, J. A. The association value of non-sense syllables. J. Genet. Psychol., 1928, 35, 255-269.
- 8. Hall, M. E. Remote associative tendencies in serial learning. J. Exper. Psychol., 1928, 11, 65-76.
- 9. Hull, C. L. Knowledge and purpose as habit mechanisms. Psychol. Rev., 1930, 37, 511-525.
- Hull, C. L. A functional interpretation of the conditioned reflex. Psychol. Rev., 1929, 36, 498-511.
- Rev., 1929, 36, 498-511.
 11. Hull, C. L. The concept of the habit-family hierarchy and maze learning. Part I. Psychol. Rev., 1934, 41, 33-54.
- 12. Hull, C. L. Goal attraction and directing ideas conceived as habit phenomena. Psychol. Rev., 1931, 38, 487-506.
- 13. Hull, C. L. The goal gradient hypothesis and maze learning. Psychol. Rev., 1932, 39, 25-44.
- 14. Hull, C. L. The concept of the habit-family hierarchy and maze learning. Part II. Psychol. Rev., 1934, 41, 134-152.
- 15. Hunter, W. S. Habit interference in the white rat and in human subjects. J. Comp. Psychol., 1922, 2, 29-59.
- Husband, R. W. A comparison of human adults and white rats in maze learning. J. Comp. Psychol., 1922, 9, 361-377.
- KLINE, L. W. An experimental study of associative inhibition. J. Exper. Psychol., 1921, 4, 270-299.
- 18. Kuo, Z. Y. The nature of unsuccessful acts and their order of elimination in animal learning. J. Comp. Psy., 1922, 2, 1-27.
- Lepley, W. M. A theory of serial learning and forgetting based uponconditioned reflex principles. Psychol. Rev., 1932, 39, 279-288.
- 20. Lumley, F. H. An investigation of the responses made in learning a multiple choice maze. *Psychol. Monog.*, 1931, 42, No. 2.
- 21. Lumley, F. H. Anticipation of correct responses as a source of error in the learning of serial responses. J. Exper. Psychol., 1932, 15, 195-205.
- 22. LUMLEY, F. H. Anticipation as a factor in serial and maze learning. J. Exper. Psychol., 1932, 15, 331-342.
- 23. MEUMANN, E. Psychology of learning. New York: D. Appleton and Company, 1913.

- 24. MITCHELL, M. B. Anticipatory place-skipping tendencies in the memorization of numbers. Amer. J. Psychol., 1934, 46, 80-91.
- 25. MUELLER, G. E., and PILZECKER, A. Experimentelle Beiträge zur Lehre von Gedächtnis. Zeitschr. f. Psychol., 1900, Ergänzungsbd., 1, 1-288.
- 26. MUELLER, G. E., and SCHUMANN, F. Experimentelle Beiträge sur Untersuchung des Gedächtnisses. Zeitschr. f. Psychol., 1894, 6, 81-190, 257-339.
- 27. PAVLOV, I. P. Conditioned reflexes. (Tr. by G. V. Anrep.) Oxford University Press, 1927.
- 28. Peterson, J. The effect of length of blind alleys on maze learning. Behav.
- Monog., 1917, 3, No. 4, Serial No. 15.
 29. Robinson, E. S., and Brown, M. A. Effect of serial position on memorization. Amer. J. Psychol., 1926, 37, 538-552.
 30. Shpola, E. M., and Israel, H. E. Habit interference as dependent upon stage of training. Amer. J. Psychol., 1933, 45, 205-227.
- 31. Spence, K. W. The order of eliminating blinds in maze learning by the rat. J. Comp. Psychol., 1932, 14, 9-27.
- 32. SWITZER, St. C. A. Anticipatory and inhibitory characteristics of delayed conditioned reactions. (To be published in the October, 1934, issue of the J. Exper. Psychol.)
- 33. Welch, G. B., and Burnett, C. T. Is primacy a factor in association-formation? Amer. J. Psychol., 1924, 35, 396-401.

Psychological Monographs

EDITED BY

JOSEPH PETERSON, GEORGE PEABODY COLLEGE

S. W. FERNBERGER, University of Pennsylvania (J. Exper. Psychol.)
W. S. HUNTER, Clark University (Psychol. Index)
H. S. LANGFELD, Princeton University (Psychol. Review)
E. S. ROBINSON, Yale University (Psychol. Bulletin)

A Classified Bibliography on Psychodietetics

BY

MARTIN F. FRITZ IOWA STATE COLLEGE

PUBLISHED FOR THE AMERICAN PSYCHOLOGICAL ASSOCIATION BY
PSYCHOLOGICAL REVIEW COMPANY
PRINCETON, N. J.
AND ALBANY, N. Y.

Science lit.
Psychology
Direct
1-20-36

TABLE OF CONTENTS

PAGI	E
A CLASSIFIED BIBLIOGRAPHY OF PSYCHODIETETICS	1
BIBLIOGRAPHY:	
Pernicious Anemia	5
Pellagra	1
Sprue	1
Acrodynia	5
Migraine	5
Epilepsy	9
Appetite	2
Racial Vigor and Temperament	5
Endurance	7
Intelligence and Learning 48	3
Mental Disorders	9
Intestinal Toxemia)
Sense Organs	1
Allergy	2
Sex Expression	3
Longevity	3
Hypertension	3
Meniere's Disease	3
Beriberi	3
Canaral 5	2

A CLASSIFIED BIBLIOGRAPHY ON PSYCHODIETETICS*

BY

MARTIN F. FRITZ

IOWA STATE COLLEGE

The relationship between diet and mental phenomena is known as *psychodietetics* and has been defined as "the science of the feeding of an individual in sickness and in health with particular reference to the mental aspect" (669).

There is now a sufficient number of reports on various phases of psychodietetics to make a comprehensive review desirable. Considerable difficulty is encountered in compiling a bibliography not only because of a tremendous literature on diet and nutrition but also because observations of psychological importance are frequently given only incidental consideration. This means that in many cases material of interest to the psychologist lies hidden in reports which are primarily concerned with biochemical and dietetic descriptions. The fact that only a few articles have appeared in strictly psychological journals indicates that psychologists have not, as yet, greatly concerned themselves with the possible relationship between diet and behavior. There is every reason to believe that an experimental attack upon the problems of psychodietetics from the psychological angle will result in much valuable information.

In general, the references have been classified according to topics which receive common attention in the journals. Articles which might be more appropriately considered under the headings of "fasting" and "drugs" have not been included.

This bibliography has been arbitrarily limited to references appearing in English up to the year 1933.

Pernicious anemia.—Considering the frequency with which mental and neurological symptoms are found in pernicious

^{*} This monograph has been subsidized by the Graduate College and the Division of Industrial Science of the Iowa State College, Ames, Iowa.

anemia, it is strange that this disease has not received more psychological consideration. Hulett (50) has emphasized the prevalence of mental disturbance and has pointed out the legal significance of this, particularly with reference to wills. It seems that mental involvement has been known for a long time and that Addison who first described the disease in 1855 mentioned the presence of mental disorders (Woltman, 123). It is common practice for investigators to attempt to distinguish between neurological and mental symptoms and while disturbances of a sensory and motor nature are nearly always reported, observations on the higher mental processes have not always been made. However, a number of writers do report observing psychical disorders in their patients (1, 2, 3, 5, 6, 9, 14, 15, 16, 21, 25, 26, 37, 51, 56, 60, 61, 62, 63, 64, 67, 77, 79, 81, 83, 85, 86, 87, 88, 90, 92, 94, 99, 100, 106, 107, 109, 111, 113, 120, 121, 122, 123, 124, 125). According to certain writers there is reason for believing that the incidence of mental difficulties associated with pernicious anemia is actually underestimated. Smith (99) thinks that mental and neurological complications are reported less frequently than they should be. Kiely (57) is of the opinion that in two of his patients, the association of neurotic symptoms and pernicious anemia was merely a coincidence although Barrett (5) feels that his experience has shown the association to be of more than incidental importance. Piney (87) and Winkelman and Eckel (120) suggest that it might be profitable to examine the blood, suspecting pernicious anemia, in all cases of mental dis-Piney thinks that when the mental symptoms have persisted for a long time, treatment will be ineffective. Barrett (6) and Woltman (123) hold that pernicious anemia plays a more important part in psychiatry than is generally supposed. Menninger (73) thinks that nervous involvement may be so slight as to be overlooked by the physician. According to Atkin (2) pernicious anemia is more often a cause of mental disease than is generally recognized. Hamilton and Nixon (46) state that the neurological examination is often poorly made or that there may even be a lack of any such examination. Murphy (79) expresses the belief that anemia is more common than is ordinarily supposed, and that neurasthenia or even mild psychoses may result from only a moderate diminution of the hemoglobin over a long period of time. Hulett (50), after a review of the reports in the literature, estimates that mental or neurological symptoms or both are found in about 90 per cent of the cases. In view of the opinions which we have cited, this estimate may be somewhat too low. This only increases the psychological significance of the malady.

From a number of reports it seems probable that neurological and mental symptoms may be present before the typical blood picture has developed (2, 10, 14, 22, 26, 29, 44, 46, 52, 54, 57, 61, 73, 88, 89, 90, 93, 113, 114, 120, 124).

According to Sturgis, Isaacs and Riddle (102) as well as Baker, Bordley and Longcope (4), the use of liver in the treatment of this disease dates from the 1926 publication of Minot and Murphy (74, 75, 76, 77). Other investigators have since found that desiccated hog's stomach is fully as efficacious as liver (13, 24, 25, 97, 103, 104, 119). Ungley (107) finds that whole brain is also effective. The influence of vitamins has received consideration (28, 31, 39, 40, 41, 58, 59, 70, 71, 72, 98, 105, 154). The question of liver extract has not been considered in this review since it belongs in the field of drugs rather than diet.

One of the interesting phases of treatment with liver or hog's stomach is the rapid mental improvement. The patient becomes brighter, feels stronger and seems to take a renewed interest in life and his own treatment (13, 47, 60, 64, 67, 92, 100, 102, 104, 110, 111, 115). There seems to be little doubt that the blood picture is markedly improved through the feeding of liver (68) but whether or not the neurological symptoms and cord lesions are likewise improved is a point over which there has been considerable controversy. A number of writers have denied that any benefit results and that at best the symptoms are merely prevented from becoming worse (1, 12, 23, 29, 30, 36, 37, 38, 52, 55, 56, 69, 82, 83). On the other hand there are a number who report objective improvement (3, 4, 8, 14, 35, 41, 42, 53, 60, 66, 67, 70, 73, 84, 92, 102, 106, 107, 108, 111, 116, 118). There are

those who report somewhat variable results and who, in general, have not taken a decided stand upon the question (32, 33, 47, 63, 76, 77, 80, 81, 87, 94, 100, 101, 102, 126). Some investigators are convinced that it takes more liver to influence the neurological symptoms than is required for the blood (7, 101, 106, 108, 112).

Return of sexual activities is mentioned by Conner (25), and

Isaacs, Sturgis and Smith (55).

Cornell (27) finds no evidence that dietary habits in the prepernicious period are the cause of the disease. Some writers have expressed the belief that pernicious anemia is a toxic condition (6, 51, 52, 65, 71, 89, 91, 94, 120, 126) although Whipple (117) doubts this. It has been suggested by Habershon (45), Herrick (48), and Scatliff (95) that emotional shock may be a causal factor. Recent evidence tends to favor deficient or defective gastric secretion (8, 18, 19, 20, 78, 96, 111, 116, 117). Pernicious anemia should be distinguished from other types of anemia (11, 17, 34, 40, 43, 45, 49, 59, 111, 112).

Pellagra.—This malady is found mostly in the Southern States and is of sufficient importance to have commanded the attention of the United States Public Health Service. Goldberger (143) estimated that in 1927 some 120,000 people in the United States suffered from an attack of pellagra. According to Wheeler and Sebrell (207) there were 7,146 deaths in 1930 recorded as due to this disease, and they estimate that there were at least a quarter of a million cases not including those of a minor degree. Cooper (133) states that pellagra as a disease entity has been known for about 200 years. However, it seems that Harris (156) was probably the first to call attention to it in America in 1902.

The literature on pellagra is filled with observations on mental symptoms and writers almost universally mention them when describing the malady (128, 129, 130, 133, 136, 138, 139, 140, 142, 148, 151, 153, 156, 157, 159, 161, 162, 163, 165, 166, 168, 170, 171, 172, 175, 177, 178, 181, 182, 183, 184, 185, 186, 189, 190, 191, 192, 193, 197, 198, 199, 203, 205, 206, 208, 211, 214). It is almost impossible to make a rational classification of the mental disorders associated with pellagra for there seems to be no characteristic pattern. The one mental symptom which could prob-

ably be considered typical is depression. Wood (214) does not think that insanity is necessarily an essential feature, though frequently present. Thurlow (198) is inclined to believe that pellagra uncovers a latent psychosis. On the other hand, there are some who believe that whatever may be the cause of the physical symptoms is also directly responsible for the mental disturbances (142, 165, 166, 183, 191, 192, 205). Langworthy (167) found upon necropsy a marked nervous degeneration. It is probably a fair interpretation to say that most writers consider the presence of mental symptoms of more than incidental importance.

That mental symptoms may occasionally precede the erythema is considered possible by Roberts (186), Shattuck (191), and Wood (214).

Nesbitt (179) finds that the economic situation, as related to food, has an effect upon the incidence of pellagra. Important differences in dietary practices between pellagrous and non-pellagrous families, as well as a seasonal variation in the supply of milk and succulent vegetables, was found by Sandels and Grady (188). Voegtlin (203) suggested that a restricted vegetable diet is the primary cause. One of the earliest etiological theories, now discarded, was that spoiled maize contained a toxic substance (129). Jobling and Arnold (164) favor intestinal intoxication, and Spies (195) mentions the possibility of a gastric disorder. Deficient diet is favored by Grimm (153), O'Leary (180), Pound (182), Vedder (201), and Warnock and Dudgeon (205), but as the only factor, is opposed by Enright (135) and Harris (158). McCollum and Simmonds (174) feel that too much weight should not be given any particular dietary constituent but that a dietary balance should be considered when interpreting the etiology of pellagra. Smith (194) believes that an abnormality of sulphur metabolism or a deficient supply of cystine is indicated by the evidence. Some form of infection is considered by Harris (158), MacNeal (177), and Viswalingam (202) to play an important part and this is the conclusion reached by the Illinois Pellagra Commission (163) in 1912. Deficient protein has been considered and Ridlon (184) finds that

pellagrins have a dietary history indicating low animal protein consumption. Guthrie (155) favors protein deficiency; Wilson (209, 210) does also and opposes the vitamin theory while Goldberger (141, 144, 147, 150) finally discarded the deficient protein theory and declared quite definitely in favor of vitamin deficiency, even naming the vitamin P-P or pellagra-preventive (142, 143, 146, 149). Greer (152) believes that a low protein allowance will result in a deficient supply of the necessary vitamin. Lustberg and Birchett (173) reject the infection theory and support vitamin deficiency. Aykroyd (127) attacks protein deficiency and is favorably disposed towards avitaminosis. Guha (154), Hindhede (160), Leader (169), and Wood (212, 213, 214) accept vitamin deficiency and this seems to be the theory most favored at present according to Thatcher (196) and Underhill (200) who have reviewed the literature on pellagra. ever, Sabry (187) opposes the theory and Bliss (131) thinks an iron rather than vitamin deficiency is indicated while Guha (154) considers iron not the sole factor but a limiting one. Regardless of the etiological theory which may be favored, treatment is nearly always dietetic (130, 132, 134, 137, 141, 145, 146, 149, 158, 165, 170, 173, 184, 185, 204, 205, 208, 214).

In 1915 Goldberger (140) attempted experimentally to produce pellagra in a group of eleven convicts and the results, he felt, justified the conclusion that it is of dietary origin. MacNeal (176) has expressed doubt that pellagra was actually produced in this experiment.

Sprue.—Until recently, sprue was considered a purely tropical disorder but Musser (224) thinks it will be found more and more prevalent in the United States as time goes on. Its similarity to pernicious anemia and pellagra has been noted by Baumgartner and Smith (218), Elders (220), Reed and Wyckoff (89), and Wood (226, 227). The general opinion seems to be that sprue should be differentiated from these two diseases.

Mental and neurological symptoms (Ashford, 215, 217; Baumgartner and Smith, 218; Musser, 224; and Reed and Wyckoff, 89) seem to be a part of the syndrome, among which have been mentioned asthenia, anorexia, "nervousness," pains in

the body, pronounced depression, sleeplessness, mental hebetude, poor memory, nightmares, neuralgias, irritability, excitability, numbness and tingling, anesthesias, paresthesias and flexor contractions.

The etiology of sprue is still in doubt. Ashford (216) suspects a mycosis superimposed upon a food deficiency or that it is a glandular deficiency due in part to an ill-balanced diet. Musser (224) doubts the etiological significance of diet but admits its value in treatment. Others (Elders, 220; Fontaine, 221; Manson-Bahr and Willoughby, 222) have great faith in dietary cures. Nye, Zerfas and Cornwell (225) conclude that Monilia psilosis is unimportant as an etiological factor. Liver has been found to be efficacious by Bloomfield and Wyckoff (219), Minot, Murphy and Stetson (223), and Manson-Bahr and Willoughby (222).

Acrodynia.—Acrodynia, also known as Swift's disease, Feer's disease, pink disease, erythredema, erythredema-polyneuritis, dermatopolyneuritis, and "raw-beef hands and feet," is a display of certain symptoms such as photophobia, paresthesia, irritability, almost incessant whining or crying, chewing of the fingers, sleep-lessness, anorexia and marked changes in disposition which certainly make it a syndrome worthy of psychological recognition. Mental and neurological symptoms occupy a prominent place in descriptions of the disease (228, 229, 231, 232, 233, 239, 240, 242, 243, 244, 249, 250, 251, 255, 258, 266).

According to Wyckoff (264), Bilderback (228) in 1920 was the first to report cases in America. The disease is probably not common (Helmick, 242), and seems to be confined almost entirely to very young children. Wyckoff (264) thinks it may occur in old or improperly nourished adults and White (261) reports two adult cases but the diagnosis is not accepted by Kernohan and Kennedy (243).

The etiology still remains obscure. Many writers (229, 230, 232, 236, 239, 240, 243, 247, 252, 253, 256, 257, 264) favor some form of infection or toxic condition in spite of a lack of definite evidence. Dietary treatment is frequently suggested (232, 237, 239, 244, 264). Favorable results have been secured through

the use of yeast (245, 248, 250, 267) and Wyllie and Stern (265) report rapid improvement when raw liver is administered. A relationship to pellagra is suggested by Bilderback (229), Weston (260), and Wood (262, 263) but rejected by Byfield (232), Emerson (235), McNeal (246), and Vipond (256). Giffen (238), Perlman (251), and Weston (259) accept some form of food deficiency as the cause while Helmick (241, 242) supports allergy. McNeal (246) rejects both food deficiency and upper respiratory tract infection. Craig (234) and Sweet (254) report relief through the use of ultra-violet light. Nesbit (248), writing in 1932, concludes that the deficiency theory is rapidly becoming the prevailing opinion. It is interesting to note that while food deficiency may not be accepted as the cause, nevertheless, dietary treatment is very commonly recommended.

Migraine.—This disorder, known since the time of Hippocrates (Rupert and Wilson, 323), has not received the psycholog.cal consideration which its symptomatology would demand. It is characterized chiefly by a periodic headache which Balyeat and Rinkel (276) report is unilateral in 94 per cent of the cases and generalized in the remainder. Other symptoms mentioned are a period of depression preceding an attack, nervousness and irritability, bulimia, druggy or profound sleep on the night preceding the headache, faintness and vertigo, scintillating scotomata, hemianopsia, photophobia, paresthesia of hands, feet or face, motor aphasia, mental confusion, nausea, emesis and exhaustion even to the extent of prostration (276, 279, 280, 296, 297, 302, 310, 313).

As to the prevalence of migraine, Balyeat and Rinkel (276) consider 7 per cent of the population a conservative estimate, while Bramwell (281) found 12 per cent of a class of senior medical students suffered or had suffered from the disorder. McClure and Huntsinger (309) present figures to show that 83 per cent of their patients developed migraine before the age of twenty. Allan (269) reports that many leading authorities believe there is an association with neuropathic taint, but he himself vigorously denies this and presents statistics to support his contention. There seems to be a very prevalent notion, sup-

ported by Ely (291), that there is some sort of relationship between migraine and epilepsy. This idea is attacked by Barborka (277), Bramwell (281), Gowers (296), Hubbell (300), and Moloney (314). Certain writers (Riley, Edinger, and Crookshank) are quoted by Allan (268) as favoring the belief that migraine is more prevalent among the cultured, professional, upper-class or brain workers, but Auerbach (268) doubts this. Allan gives figures to show that there is no relation to occupation. Riley (317) and Rupert and Wilson (323) think that considerably more women than men are affected but Allan (270, 272) concludes there is no sex difference in the number of sufferers although there is a difference in intensity. Buchanan (286) thinks there is no harmful influence on longevity. Allan (271) finds that more than half of the patients have an attack once in two weeks or oftener.

Many theories have been advanced on the etiology of migraine (Gordon, 294). Bramwell (281) believes that anxiety and worry are important in precipitating an attack. An experimental demonstration of a poison in the blood is reported by van Leeuwen and Zeydner (307). Hurst (303) finds no evidence in favor of a gastrointestinal toxemia but Hartsock (298) accepts this explanation. Carbohydrates are suspected by Brown (283, 284, 285) and Minot (312). The ketogenic diet is favored by Barborka (277, 278), Pollock and Barborka (315), and Schnabel (324). Dietary measures, in the experience of Kennedy (304), never cure but do reduce the frequency of attacks. Although allergy is denied by Hartsock (298), Hartung (299), and Stevens (326), there are many writers who very definitely favor some form of sensitization as an explanation of the cause (273, 274, 275, 276, 279, 282, 283, 284, 285, 287, 288, 289, 290, 292, 305, 306, 308, 309, 311, 314, 316, 318, 319, 320, 321, 322, 325, 328, 329, 330).

Epilepsy.—It is not necessary to emphasize the psychological importance of epilepsy because the profound and characteristic mental symptoms are already well known (335, 336, 339, 344).

Estimates on epilepsy show that there must be some hundreds of thousands of cases in the United States. Allan (269) agrees

with Clark (342) who gives 0.20 to 0.33 per cent of the general population. Robertson (382) gives 235,000 cases and Davenport (353) reports 5.15 per thousand for 2,500,000 drafted men during the World War. Barborka (331) thinks 500,000 is a conservative estimate.

Collier (351) and Robertson (381, 382) consider epilepsy to be caused by a toxic condition. A number of writers have proposed allergy as a cause (290, 352, 363, 365, 366, 371, 383, 384, 387, 388, 395, 397, 398, 399). Allergy is rejected by Cohen and Lichtig (350), Felsen (354), Lennox and Cobb (364), Moloney (314), and Smith (386). Wilder (402) was probably the first to report on the value of the ketogenic diet in reducing or controlling epileptic seizures and this method of treatment has received considerable support (331, 332, 333, 334, 337, 338, 357, 358, 359, 360, 361, 362, 364, 368, 373, 374, 375, 376, 377, 378, 379, 386, 389, 390, 391, 392). Bridge and Iob (337) suggest that the ketogenic diet is effective due to the removal of surplus extracellular fluid from the body but Cameron (340) presents evidence to show that dehydration by means of a low water content diet has no definite effect upon the number of seizures. The most favorable results are secured with the ketogenic diet in cases of petit mal, according to Peterman (375), Higgins (362), and Pulford (379). Barborka (333, 334), Fetterman (355), Higgins (362), Shanahan (385), and Smith (386) urge early treatment before seizures have become fixed and it seems necessary to enforce the diet rigidly (Barborka, 332; Harper, 358; Henderson, 361; and Shanahan, 385) since the patients will often secretly obtain other food, particularly carbohydrates. Rowe (319) believes that the ketogenic diet is effective partly because foods to which the subject may be sensitive, such as wheat and milk, are removed. Tracy (393) urges dietary treatment but does not make specific recommendations other than a plentiful supply of vitamins. Lennox and Cobb (364) doubt the value of protein McMurray (367) restricted carbohydrates, along with other therapeutic measures, and found improvement. Walker and Wheeler (396) found that convulsions decreased on a pellagra-producing diet. Weeks, Renner, Allen and Wishart (400)

tried various special diets and came to the conclusion that they were neither harmful nor beneficial. Although Clark has been one of the strongest exponents of the theory that epilepsy is psychogenic (343, 344, 346, 347, 349) and suggests reëducation (345) in its treatment, he is also favorably disposed toward a low protein diet (348) and has urged dietetic treatment (341). Just how the psychogenic theory, also defended by other writers (Fox, 356; Marsh, 369; Martin, 370; Orbison, 372; Richmond, 380; Turner, Read et al., 394; Wiersma, 401; and Wilson, 403), will be harmonized with the apparent success of the ketogenic diet, remains to be seen.

Appetite.—As early as 1911, Osborne and Mendel (461) expressed the belief that it was not monotony but some deficiency in the diet which brought on anorexia, and this has been confirmed experimentally in recent years. It now seems to be clearly established that a deficiency in vitamin B complex will cause a loss of appetite (409, 414, 415, 416, 417, 418, 419, 420, 423, 424, 425, 431, 433, 436, 438, 439, 443, 448, 463, 468, 470, 471, 472, 473, 475). Sherman and Sandels (468) report that lack of vitamin B (B₁) is more responsible for anorexia than is lack of vitamin G (B₂). Wright (475) thinks the evidence indicates that a direct effect of vitamin B deficiency upon the alimentary tract is responsible for the loss of appetite.

Allison and Davies (404) recommend dietetic treatment to intensify appetite in functional cases and Bartlett (405) has found that fresh calf's liver is beneficial. Twenty-four per cent of 1,471 children coming to the hospital were found by Bartlett to have loss of appetite as a primary symptom. Schultz (466) is opposed to the use of large quantities of cow's milk in a child's diet and finds that its removal aids in overcoming anorexia. Kerley (441) thinks that hyperchlorhydria is a frequent cause of defective appetite in children and that refusal of the breast may be caused by a mental shock to the mother (440). Berkman (407) considers anorexia nervosa but doubts that it is so very common. Kugelmass and Samuel (444) believe that psychic discord will disturb the functions of the alimentary tract which will cause loss of appetite.

That raw vegetables are preferred to cooked ones by children and that a marked desire for food seems to develop gradually are conclusions reached, on the basis of an investigation, by McLaughlin, Tarwater, Lowenberg, and Koch (454). Food dislikes acquired in childhood are thought by Rice (464) to have an influence upon metabolic disorders of middle life. Young (476, 477) has developed a method of studying objectively the preferences of rats and finds that they prefer in order milk, sugar, butterfat, wheat and flour. Levine (445) states that the Eskimo actually dislikes sugar and gets used to salt more quickly than to sugar. Gauger (429) found that preschool children will modify their responses to an apparently disagreeable taste stimulus. Holder, Smith, and Hawk (435) conclude that utilization is not affected by palatability. McCollum (452) found that by giving attention to the palatability of food the decrease in body weight of rats could be deferred but not permanently stopped.

Instinct as a guide to food has been speculated upon a great deal and has also received some experimental consideration. A number of writers favor the idea that there will be a relatively correct choice of diet when the animal is presented with a variety of foods, some authors postulating an instinct while others suggest some sort of chemotropism (406, 408, 412, 418, 421, 422, 424, 427, 432, 434, 437, 447, 449, 450, 451, 456, 457, 459, 460, 462, 465, 474). In 1906, Fisher (428) performed an experiment on "natural eating" and found a voluntary reduction in the consumption of flesh foods. However, Chittenden (413), Kon (442), Levine (445), McCollum, Simmonds, and Pitz (453), and Nevens (458) doubt that any innate craving or appetite can be safely relied upon. Slonaker and Card (469) observed increased cannibalism in rats when the supply of animal protein was reduced, which they attribute to a craving for animal protein. Sherrington has implied that the aversion of dogs for dog meat is nearly universal, but Girden (430) and Maslow (455) do not find this to be the case.

Racial Vigor and Temperament.—An attempt has been made to explain racial and national differences on the basis of diet. The idea seems to be quite prevalent that the less vigorous

people, lacking initiative and energy, are vegetarians. Chinese and Japanese, as well as other Orientals, are frequently referred to in order to support this hypothesis (Adolph, 478; Armitage, 479; DuBois, 486, Lorand, 496). Chittenden (413) quotes Oshima (510) as saying that the Japanese rural population of the interior eats fish but once or twice a month and meat almost not at all. This is in disagreement with Hirai, quoted by Holt (492) as stating that the Japanese "rarely get meat but take much fish." However, Oshima writes that there have been too few investigations to warrant a definite conclusion on the national diet. Chun (483) suggests that a small amount of animal protein and deficiency of fat-soluble vitamin are responsible for the lack of energy among the Chinese poorer classes. Benedict (406) considers that productive power, enterprise and civilization are associated with liberal quantities of protein. Hitchcock (489, 490) has studied the effect of protein upon voluntary activity of the white rat and finds that a diet containing 12 per cent protein of vegetable origin depresses activity. Slonaker (512, 513, 514) also found that a vegetarian diet so much decreased the activity of the white rat that it resembled senile conditions. Conger's (484) investigation showed that rats on a well balanced diet voluntarily took more exercise than those on diets either lacking animal protein or containing a very large amount. Tang, Chin, and Tsang (516) studied the effects of a vegetarian diet upon learning ability in rats. Wu and Wu (526) conclude that no adequate vegetarian diet for the white rat is known at present. McCollum, Simmonds, and Parsons (500) favor a liberal amount of protein in the diet but believe that it is dairy products rather than meat which is responsible for racial vigor. Low efficiency, reduced morale, loss of endurance, fatigability, tendency to forget and loss of initiative among the people of Germany during the World War are attributed by Mason (502) to a low protein diet. Hindhede (160) favors a vegetarian diet with but a small consumption of meat.

It has been stated that animals fed a meat diet become vicious and dangerous while a vegetable diet will make them tame and easily trained (Liebig, 495; Lorand, 496; and Carpenter, 412).

These characteristics of viciousness and docility are likewise supposed to vary with diet in human beings. However, DuBois (486) calls attention to the fact that the meat-eating Eskimo is perhaps the most peaceful and unwarlike race in the world. and Anderson lived on an exclusive meat diet for a year without any apparent ill effects and no decrease in mental vigor was observed (Lieb, 493, 494; and McClellan, 498). Miles (481) found that the psychological effects of a limited food consumption were, in general, rather small. Although productivity and initiative have often been ascribed to the type of food consumed (Berman, 408; Grayson, 487; and Murlin, 508), it seems that Sherman (511) and Wherry (522) have been among the few bold enough to challenge this hypothesis. Wherry points out that with the high incidence of beriberi in Japan and hookworm in India, we may well question whether or not a low protein diet is the cause of racial inferiority. When vegetarianism is voluntarily assumed, it is remarked by Mendel (504) that it generally involves a moral aspect rather than a physiological basis.

Berman (408), in discussing the food habits of leaders, calls attention to the enormous meals consumed by Bismarck, exponent of the "blood-and-iron" policy, and Penrose, political boss of Pennsylvania, as contrasted with the abstemiousness of Gladstone and Mahatma Gandhi. Crile (485) suggests that a hyperkinetic drive may be due to autointoxication (foreign proteins) and excessive diet. Trembly (518) observed that white rats on a ketogenic diet were considerably more active than the control animals. Weston (521) reports that with vitamin C deficiency there may be observed a change in disposition. Stern (515) expresses his belief in a relationship between diet and personality.

The specific dynamic action of food, especially protein (480, 486, 497, 507, 524), and metabolism in relation to the psychological situation has received some consideration. Whitacre and Blunt (523) investigated the relationship of temperamental type to the dynamic effect of food. They were unable to show any difference between vivacious and placid individuals. It has been observed that Orientals possess a lower basal metabolism than Occidentals (501, 503, 506, 509) even when a correction is applied

to the usually accepted standards for women. Heinbecker (488) has shown that the basal metabolism of the Eskimo is about one-third higher than for people living in temperate zones. Wakeham and Hansen (519, 520) find that long-time vegetarians have a somewhat lower basal metabolism while Wu and Chen (525) find the values for vegetarian rats somewhat below those of omnivorous rats. Benedict and Roth (482) doubt that a vegetarian diet alters basal metabolism.

It has been McCollum's (499) experience that white rats may not show any immediate effects of certain mildly unfavorable diets but there will be cumulative effects so that the strain dies out after three or four generations. Minot (505) has expressed his belief in such cumulative effects in the case of human beings. Whether or not such physical deterioration through several generations is also accompanied by mental deterioration has not been specifically investigated.

Endurance.—Considerable attention has been given the possibility that the amount of protein in the diet is a factor in endurance. Berry (528), using a two mile run as a test of ability to resist fatigue, found that results always favored the low proteid group. The investigation was carried on for a period of four months. According to Chapin (529), a reduction in proteid food will, in a few days, permit a greater output of work, but Bassett, Holt, and Santos (527) found no demonstrable effect upon physical capacity when meat was withheld from the diet for a period of a week. Fisher (428, 530, 532) concluded on the basis of experimental evidence that a low-proteid, non-flesh dietary was a decided advantage from the standpoint of endurance. He has also called attention to a Belgian experiment which was found to support his results (531). Hindhede (533, 534) reports that men on a low vegetable protein diet have unusual capacity for work and endurance. Grayson (487) is of the opinion that a vegetarian diet increases endurance. Howe (535) observed the effects of high and low protein diets on ergographic responses. He concluded that the advantages were essentially in favor of a low protein diet. Thompson (542) opposes a strictly vegetable diet on the ground that it induces muscular weakness and languor. In his opinion, animal food in some form is absolutely essential to vigor. The loss of efficiency and loss of endurance, among other effects noted in Germany during the war, are attributed by Mason (502) to the low protein diet. Langworthy (538) reports that four out of six successful contestants in a strength and endurance test, who later furnished data, had lived upon an ordinary mixed diet. It should be pointed out that the complications in the problem of endurance as related to protein, apparently have not always been clearly realized. The question of animal vs. vegetable protein may be quite distinct from the question of a high vs. low protein diet.

Berman (408) describes the sustaining effects of sugar in the case of marathon runners. Lorand (496) agrees that sugar possesses counter fatigue effects while Laird (537) reports experimental confirmation of this fact. Krogh and Lindhard (536) found that four out of six subjects experienced, subjectively, less fatigue on a high carbohydrate diet than on a high fat diet.

Nelson and others (539) have shown that the absence of vitamins A and B in the diet of the white rat will result in lessened muscular endurance. A clinical case is reported by Peters (540) where an increase in vitamin B in the diet caused a rapid disappearance of listlessness and fatigability. Talbot (541) mentions poor food habits and malnutrition as one of the many causes of fatigue. Miles (481), in a study of reduced food intake, found no objective effects but, subjectively, there was a distinct tendency to report lowered endurance and greater susceptibility to fatigue. Holck (491) reports that "Fletcherizing" decreased muscular endurance.

Intelligence and Learning.—Considering the popular belief in a close relationship between the state of nutrition and mental ability, it is strange that so few experiments have taken up this question. Levine (562) believes that malnutrition has marked psychological effects. Graper and Park (557) and Holt (492) are on record as favoring some connection between mental development and nutrition. McCollum (563) believes that a very poor diet will dull the mental capacity of children and warp their personalities. This viewpoint is opposed by other writers.

Blanton (547), after an extended study of malnourished children in the immediate war area of Germany, came to the conclusion that very few had suffered any permanent impairment of intelligence, although lack of energy and marked susceptibility to fatigue were noticeable. He also failed to find any definite increase in the number of children suffering from neuroses and psychoses. Dowd (551) reports that even under nutritional care the I.Q. remained substantially the same and this is supported by Smith and Field (575). Rosenberg (573) failed to find any appreciable difference in I.Q. (Stanford-Binet) between underweight children on a meatless and eggless diet as compared to those on a "representative American dietary," after six months of such treatment. Nicholls (571) found that underweight children were inferior to normals on certain tests involving essentially a muscular situation, but there was no difference on tests similar to those used in intelligence tests. Hunt, Johnson, and Lincoln (560), employing a number of psychological tests, found that underweight children actually had a slight advantage. Stalnaker (576) calls attention to the fact that height and weight may not be satisfactory standards for judging malnourishment. Although experiments have met with little success in showing a direct effect of malnourishment upon intellectual capacity, the question still has considerable importance since achievement may be lowered through lassitude and ill health. Levine (561) presented statistics in 1925 showing that at least four million school children were suffering directly from malnutrition. Bliss (548) found in his study that a fourth to a third of the children were underweight and therefore assumed to be malnourished. Cramer (549, 550) thinks that many people are in a chronic state of vitamin underfeeding and that a lasting weakness may in this way be carried over from infancy.

There is no permanent effect of rickets upon mental development, according to Gesell (556). Peters (572) thinks that there is a noticeable association of rachitis and idiocy. Frank (554) was unable to demonstrate a difference between normal and rachitic rats, although the normals were superior in a relearning test three months later. Anderson and Smith (543, 544) found

that qualitatively and quantitatively stunted rats were superior to those on a normal diet in relearning a maze, but in a second relearning period, following realimentation, they tended to become similar. Using the escape-from-water technique, Ruch (574) showed that young rats on a maintenance diet were superior to those on either a restricted or liberal diet. Pearson (553) is quoted as stating that "health and intelligence are correlated, although not very markedly." Balyeat (545, 546) finds that allergic children are definitely superior to non-allergic in mental activity. An investigation by Hoefer and Hardy (558) shows that breast feeding, when not continued too long, has a favorable influence upon the intellectual ability of children.

The possibility that vitamin B may have some influence upon intelligence and learning ability has been considered. Maurer and Tsai (566, 567, 569) have given this problem experimental consideration and report that lack of vitamin B in nursing rats will result later in a decided effect upon learning ability. However, their generalizations (565, 568) have been very severely attacked (552). Dennett (423) believes that cow's milk, and in some cases human milk, does not contain the optimal quantity of vitamin B. Hoobler, Outhouse, and Macy (559) and Macy, Outhouse, Graham, and Long (564) find that human milk is often deficient in vitamin B, while Moore, Brodie, and Hope (570) find the same thing true in the case of the rat. Fritz (555) did not find that the accuracy of maze performance was affected by a vitamin B-deficient and salt-defective diet in relatively mature rats.

Miles (481) reports that no evidence was found of lessened ability to do college work while on a quantitatively restricted diet.

Holck (491) found that "Fletcherizing" had no significant effect upon sleeping time, mental multiplication and typewriting speed but that typewriting accuracy was reduced.

A vegetarian diet was found by Tang, Chin, and Tsang (516) to have an unfavorable influence upon the learning of male rats but was without effect in the case of females.

Mental disorders.—The term "mental disorders," for purposes of this review, is given a liberal interpretation to include

not only psychoses and neuroses but also so-called nervousness. A variety of references not readily classified under some other topic are here considered.

Fitch (583) discusses the dietary peculiarities which may result from mental abnormality. Levine (562) is convinced that malnutrition will produce marked anatomical changes in the nervous system, and that there will be emotional disturbance, irritability, melancholia and apathetic behavior. On the basis of clinical evidence, Mercier (591) concludes that mental disease, in at least a few cases, is due to error in the diet. Walsh (598, 599) also is of the opinion that many patients suffering from neurotic and psychoneurotic symptoms are only in need of dietary treatment. Buckley (581) considers diet of importance in the treatment of neurasthenia, while Paton (592) thinks our ignorance of the true nature of neurasthenia, as well as metabolism, makes it impossible to formulate any laws for dieting in this situation. Grayson (487) and Lorand (496) believe that a vegetarian diet reduces nervousness. In the opinion of Holt (492), most of the neuroses of childhood depend entirely upon disorders of nutrition. Seham and Seham (594) find that forced feeding and rest cure for malnourished children reduces nervous symptoms. Laird, Levitan, and Wilson (589) present experimental evidence to show that milk given to school children at 9:30 A.M. Hartwell and Mottram (585) find that reduces nervousness. the feeding of brown bread causes rats to become highly nervous and they question the propaganda in favor of brown bread. Bryant (580) considers diet an important factor in the treatment of hyperkinetic individuals. Henry (586) and Richardson (593) are convinced that there is an intimate relationship between emotional disorders and digestive functions. Bostock (578) reports achlorhydria and hypochlorhydria to be surprisingly frequent in the psychoses. Delirium tremens may be successfully handled through dietary measures according to Carter, Howe, and Mason (582). Bronfenbrenner (579) calls attention to the great craving of the feebleminded for carbohydrates. In contrast with the above findings, we have Appel and Farr (577) and Sherrill (595) expressing some doubt that diet is a cause of mental disorders.

Gregg (584), calling attention to the statistics on depression and suicide, makes the interesting suggestion of a connection with vitamin shortage. McCarrison (590, 450) has shown experimentally that brain weight is markedly increased by a diet excessively rich in starch and fat but lacking in vitamin B. He suggests a relationship to mental disorder. Hughes and others (587, 588) have shown that lack of vitamin A results in nervous degeneration in swine and that chickens, cows and pigs will develop incoördination and spasms. Thomas (596) states that vitamin D is valuable in bringing about a reversal of the hemoclastic crises in dementia praecox. Timme (597) reports very little success with the use of calcium mobilizing agents involving vitamins B and D in an attempt to overcome hypocalcemia in patients who were decidedly hyperirritable.

Intestinal toxemia.—The belief seems to be quite prevalent that intestinal toxemia will lead to profound mental disturbances and since its treatment is generally dietetic, it becomes necessary to consider this subject in a review on psychodietetics. Bartle (602) lists certain symptoms which are of psychological significance, such as fatigue, nervousness, sleeplessness, melancholia, anorexia, nausea, prodromal well being, bulimia, drowsiness, headache, migraine, phobias, vertigo, mental hebetude, and vomiting. Mental disturbances are mentioned by other writers (Boles, 603; Cotton, 604; Craig, 352; Crile, 605; Diamond, 606; Fitch, 609; Kauffman, 612; Kraetzer, 615; Satterlee, 617; Satterlee and Eldridge, 618; Stucky, 620; Walker, 622; Wile, 623; and Yearsley, 624).

Alvarez (600), in a review on the subject, leaves the impression that sound scientific knowledge on intestinal toxemia is scarce and that the effects have been somewhat overstated. Andrewes (601) thinks there is little evidence that toxins derived from the ordinary flora of the intestinal tract are important in alimentary toxemia. Fitch (608), in an earlier publication, announced an "open mind" on the subject, while Gant (610) and Wile (623) are somewhat dubious concerning the severe mental effects of

constipation. However, it should be pointed out that most writers differentiate between constipation and intestinal toxemia, although recognizing a close relationship. Gross (611) considers it proved that lack of vitamin B will lead to intestinal stasis. Kellogg (613, 614) has been most vigorous in affirming the evils of constipation. According to Synnott (621), there is no relation between intestinal putrefaction and constipation. Paulsen (616) attacked the problem of intestinal toxemia experimentally and treated a group of 30 girls by dietary methods, adequate elimination, exercise, hydrotherapy, and a cheerful environment. On a large number of psychological tests, the experimental group was found to be quite decidedly superior to an untreated control group, although the difference was greater for motor reactions than in the case of intellectual processes. Donaldson (607) observed five men who engaged in voluntary constipation for a period of nearly four days. A number of symptoms of psychological interest appeared but he concludes that these are to be explained on a mechanical basis, rather than toxic, since they disappeared so quickly after an evacuation by means of an enema.

Allergic toxemia is recognized by Rowe (321) who lists as symptoms drowsiness, mental confusion, slowness of thought, lack of initiative and ambition, irritability, despondency, fatigue, weakness, bodily aching, and a feeling of "being poisoned."

Sense organs.—Nightblindness is perhaps the most prominent sensory disorder of psychological importance associated with diet. Adler (625) has briefly reviewed the literature on nightblindness and Bordley (630) has summarized the controversy over the terminology. Aykroyd (626, 627) calls attention to the fact that this disorder has been known far back into antiquity and that the cure was also known, viz., the eating of liver. Little (634), as well as Aykroyd, has observed much nightblindness among the people of Newfoundland and Labrador. Spence (638) reports that occasional cases due to dietary deficiency are to be seen in the industrial districts of northern England. Enright (631) quotes Keatinge as stating that there was an extraordinary prevalence of nightblindness among native troops in Egypt during

the war. Pillat (636) mentions the malady as one of the eye diseases encountered in Chinese soldiers suffering from vitamin A deficiency. It seems to be well established that lack of vitamin A is the cause (Bloch, 629) and the experimental work, as well as clinical results, indicates this to be correct. Fridericia and Holm (632) found that lack of vitamin A along with exposure to strong light caused a delay in the regeneration of visual purple in the retinae of rats. They were not able to show this when vitamin B was withheld from the diet. Holm's (633) experiments show that the disorder appears very early during the avitaminosis. Tansley's (640) experiments also showed a lowered rate of regeneration of visual purple in white rats fed a vitamin A deficient diet. Smith (637) distinguishes between nutritional nightblindness and idiopathic nightblindness, generally called retinitis pigmentosa.

Peterson (635) mentions that cattle will become blind in three months when fed cotton seed cake. Spivacke (639) reports a case of temporary blindness resulting from an allergic reaction to mustard given as a test for hypersensitivity.

Barlow (628) was unable to show that rickets is a factor in deafness. Weston (641) states that an excess of vitamin B over vitamin A will cause a proliferation of lymphoid tissue sufficient to block the eustachian tubes and interfere with hearing.

According to Weston (641), vitamin C deficiency causes minute hemorrhages into the taste buds, resulting in an absence or perversion of taste.

Allergy.—Certain references to allergy receive consideration under the headings of migraine (273, 274, 275, 276, 279, 282, 283, 284, 285, 287, 288, 289, 290, 292, 298, 299, 305, 306, 308, 309, 311, 314, 316, 318, 319, 320, 321, 322, 325, 326, 328, 329, 330), epilepsy (290, 314, 319, 350, 352, 354, 363, 364, 365, 366, 371, 383, 384, 386, 387, 388, 395, 397, 398, 399), intelligence (545, 546), intestinal toxemia (321), sense organs (639), and Meniere's disease (662). The frequency with which mental symptoms are mentioned in connection with hypersensitivity makes this topic of some importance in psychodietetics.

Duke (644) estimates that about 12 to 15 per cent of the

people are hypersensitive while Rowe (648) considers the figure to be about 30 per cent.

Talbot (653) thinks that the findings on allergy are of great importance. Rowe (320, 649) enumerates symptoms such as confusion, restlessness, irritability, fatigue, dizziness, drowsiness, weakness, nervousness, dullness, and generalized aching. Carr (643) reports the development of headache, nausea, and drowsiness in a patient whenever he was placed on a milk diet. Sherwood-Dunn (652) feels that anemia, neurasthenia, migraine, insomnia, and melancholia are associated with anaphylaxis. Shannon (651) is convinced that anaphylaxis causes nervousness through irritation of the nervous system and that this is also the correct explanation of the so-called neuropathic diathesis. Craig (352) has observed epileptic fits and mental confusion follow the ingestion of eggs and has seen three cases of delirium follow the taking of mushrooms. Andresen (642) thinks it is highly probable that many of the symptoms usually attributed to intestinal toxemia are really due to food sensitivity.

Miller (371) calls attention to the fact that until comparatively recently asthma was considered a neurosis (Gerrish, 646) and even at present many physicians emphasize the importance of nervous phenomena. It seems to be well established that a certain number of patients can be relieved by removing offending foods from the diet (Maisel, 647). Duke (645), who is one of the exponents of allergic theory, has observed mental abnormalities associated with asthma.

Sheard, Caylor, and Schlotthauer (650) found that guinea pigs, white swine and goats displayed photosensitivity after the ingestion of buckwheat. Guinea pigs showed the most marked effects with shaking of the head, excitation, squeaking, scratching of the ears, and intense agitation. Darkness calmed them. No effects were observed in rabbits, dogs, white mice, and white rats.

Sex expression.—It would seem to be probable that sex expression is subject to dietary influence. Miles (481, 656) reports decreased sex interest and activity for 22 out of 24 men who had been placed on a restricted diet. He concludes that a

relatively high metabolic level is necessary for the normal functioning of sex. When male rats are placed on a diet lacking vitamin E they eventually lose all sex interest, according to Evans (654, 655). Fritz (555) was unable to show any sex difference in the maze performance of white rats while on inadequate diets but did find that a significantly greater number of the males survived than the females. Tang, Chin, and Tsang (516) found some sex differences in rats learning a water maze while on a vegetarian diet. Mason (502) writes that due to malnutrition in Germany during the war, there was suppression of menstruation for long periods of time in women and a greatly decreased sexual libido in the men.

Longevity.—Longevity can be significantly improved through enriching the diet, according to the experiments of Sherman and Campbell (657). Slonaker (659) has found life span to be a function of the amount of protein in the diet, the optimum being 14 per cent. A liberal allowance of vitamin A increases length of life considerably, according to Sherman and MacLeod (658).

Hypertension.—Jump (660) reports fatigability, vertigo, lack of initiative, nervous instability, dyspnea, and headaches as symptoms of essential hypertension. He recommends complete rest, a milk diet and the avoidance of foods which disagree with the patient. The elimination of proteins is generally recommended although their effect upon blood-pressure is still undecided. Rowland (661) mentions that headache, neuralgias, paresthesias, vasomotor instability, palpitation, insomnia, and nervous irritability accompany hypertension and advocates a balanced reducing diet as the most important treatment.

Meniere's disease.—This disease may be caused by food allergy, according to Duke (662). In describing this disease, Kopetzky (663) emphasizes the sudden onset of deafness, tinnitus and vertigo, and states that other writers have reported loss of consciousness, double vision, transitory clouding of the visual fields, transitory hemiopia, psychic depression, and loss of memory. Richey (664) reports that improvement has been noted when meat is withdrawn from the diet.

Beriberi.—It is quite universally accepted that beriberi, caused

by a deficiency of vitamin B (F or B₁), will result in peripheral nerve degeneration (Vedder, 667; Zimmerman, 668). Sensory and motor disturbances are characteristic of this disease (Lovelace, 666; Vedder, 667) among which will be loss of deep reflexes, pains in the legs, "pins-and-needles" sensations, paraplegia, peculiar "stiff" gait, anesthesia, paresthesia, and absent or exaggerated kneejerks. It seems there is little evidence that the higher mental processes are affected in beriberi even though Funk (665) has presented evidence which suggests a breakdown of the lipoids of the brain. Farnell and Yacovlev (136) report mental depression at the onset and afterwards indifference accompanied by mental prostration. However, they warn that mental symptoms are not so characteristic as the neurological symptoms, and oftentimes may be absent. It is their opinion that beriberi and pellagra rarely exist independently of each other, which calls attention to the close association of vitamins B and G (B₁ and B₂). Hoobler (436) writes that children suffering from infantile beriberi become restless, fretful, and develop a feeble, plaintive cry as well as displaying neurological symptoms.

BIBLIOGRAPHY

Pernicious Anemia

- Ahrens, R. S. Neurologic Aspects of Primary Anemia. Arch. Neurol. and Psychiat., 1932, 28, 92-109.
 Atkin, I. A Case of Pernicious Anemia Associated with Mental Disease.
- Lancet, 1932, 2, 569.
- 3. BAKER, B. M., BORDLEY, J., and LONGCOPE, W. T. The Effect of Liver and Liver Extract upon the Symptoms and Signs Referable to the Nervous System in Pernicious Anemia. Minnesota Med., 1930, 13, 815-817.
- 4. BAKER, B. M., BORDLEY, J., and LONGCOPE, W. T. The Effect of Liver Therapy on the Neurologic Manifestations of Pernicious Anemia. Amer. J. Med. Sci., 1932, 184, 1-24.
- 5. BARRETT, A. M. Mental Disorders and Cerebral Lesions Associated with Pernicious Anemia. Amer. J. Insanity, 1913, 69, 1063-1078.
- 6. BARRETT, A. M. Mental Disorders Associated with Pernicious Anemia. Fifth Biannual Report, State Psychopathic Hosp., Univ. of Michigan, biennial period ending June 30, 1916.
- 7. Beebe, R. T., and Lewis, G. E. The Maintenance Dose of Potent Material in Pernicious Anemia. Amer. J. Med. Sci., 1931, 181, 796-812.
- 8. BIERRING, W. L. Liver Therapy in Pernicious Anemia. J. Iowa State Med. Soc., 1927, 17, 367-370.

- 9. BILLINGS, F. The Changes in the Spinal Cord and Medulla in Pernicious Anemia. Boston Med. and Surg. J., 1902, 147, 225-233, 257-263.
- Bramwell, B. Remarks on a Case of Subacute Combined Degeneration of the Spinal Cord, Simulating Disseminated Sclerosis. Brit. Med. J., 1910, 1, 1396-1398.
- 11. Brill, I. C. The Specificity of the Minot-Murphy Diet in Pernicious Anemia. J. Amer. Med. Assoc., 1927, 89, 1215.
- 12. Brockbank, W. Discussion on the Value of Liver in Treatment. Proc. Royal Soc. Med., 1931, 24, 951.
- 13. Brower, A. B., and Simpson, W. M. Antianemic Influence of Desiccated Whole Hog Stomach. Amer. J. Med. Sci., 1931, 182, 319-326.
- 14. Bubert, H. M. Subacute Combined Sclerosis. J. Amer. Med. Assoc., 1928, 90, 903-906.
- 15. CADWALADER, W. B. Diagnosis of Subacute Combined Sclerosis of the Spinal Cord Associated with Severe Anemia. J. Amer. Med. Assoc., 1916, 66, 2035-2036.
- 16. CAMP, C. D. Pernicious Anemia Causing Spinal Cord Changes and a Mental State Resembling Paresis. Med. Rec., 1912, 81, 156-158.
- 17. Cantarow, A. Progress in Medicine. Internat. Clin., 1932, 1, 42nd series, 190-250.
- 18. Castle, W. B. Observations on the Etiologic Relationship of Achylia Gastrica to Pernicious Anemia. I. The Effect of the Administration to Patients with Pernicious Anemia of the Contents of the Normal Human Stomach Recovered After the Ingestion of Beef Muscle. Amer. J. Med. Sci., 1929, 178, 748.
- CASTLE, W. B., and TOWNSEND, W. C. II. The Effect of the Administration to Patients with Pernicious Anemia of Beef Muscle After Incubation with Normal Human Gastric Juice. Amer. J. Med. Sci., 1929, 178, 764.
- 20. Castle, W. B., Townsend, W. C., and Heath, C. W. III. The Nature of the Reaction Between Normal Human Gastric Juice and Beef Muscle Leading to Clinical Improvement and Increased Blood Formation Similar to the Effect of Liver Feeding. Amer. J. Med. Sci., 1930, 180, 305-335.
- 21. CHRISTIAN, H. A. Pernicious Anemia. Internat. Clin., 1922, 1, 32nd series, 8-14.
- 22. CLARKE, J. M. On the Spinal Cord Degenerations in Anaemia. Brain, 1904, 27, 441-459.
- 23. Cohen, A. E. Subacute Combined Sclerosis Progressive During Remission of Pernicious Anemia. J. Amer. Med. Assoc., 1928, 90, 1787.
- 24. Conner, H. M. The Treatment of Pernicious Anemia with Swine Stomach. J. Amer. Med. Assoc., 1930, 94, 388-390.
- 25. Conner, H. M. The Feeding of Gastric Tissue in the Treatment of Pernicious Anemia. J. Amer. Med. Assoc., 1931, 96, 500-503.
- 26. CORNELL, B. C. Hydrochloric Acid as a Prophylactic Measure Against Pernicious Anemia. J. Indiana Med. Assoc., 1929, 22, 104.
- 27. Cornell, B. S. A Study of the Pre-disease Diets of Patients with Pernicious Anemia. Bull. Johns Hopkins Hospital, 1927, 40, 409-421.
- 28. Cramer, W. On the Mode of Action of Vitamins. Lancet, 1923, 1, 1046-1050.
- 29. Curschmann, H. Nervous Disturbances in Pernicious Anemia and Liver Therapy. Arch. Neurol. and Psychiat., 1931, 25, 438-439.
- 30. Davison, C. Subacute Combined Degeneration of the Cord. Arch. Neurol. and Psychiat., 1931, 26, 1195-1219.

- DAVIDSON, S. Vitamin B in Anaemia. Lancet, 1931, 2, 1395-1398.
 DAVIDSON, S., McCrie, J. G., and Gulland, G. L. The Treatment of Pernicious Anaemia with Liver and Liver Extracts. Lancet, 1928, 1, 847-852.
- 33. DYKE, S. C. Discussion on the Value of Liver in Treatment. Proc. Royal Soc. Med., 1931, 24, 935-948.
- 34. ELLIOTT, C. A., NADLER, W. H., and STARR, P. Liver Feeding in Pernicious Anemia. Med. Clin. North Amer., 1927, 11, 147-155.
- 35. FARQUHARSON, R. F., and GRAHAM, D. Liver Therapy in the Treatment of Subacute Combined Degeneration of the Cord. Canadian Med. Assoc. J., 1930, 23, 237-244.
- 36. Fried, B. M. Subacute Combined Degeneration of the Spinal Cord in Pernicious Anemia. J. Amer. Med. Assoc., 1929, 92, 1260-1263.
- 37. GARVEY, P. H., LEVIN, P. M., and GULLER, E. I. The Effect of Liver Therapy on the Neurologic Aspects of Pernicious Anemia. Ann. Int. Med., 1933, 6, 1441-1448.
- 38. Gibson, R. B., and Fowler, W. M. Effects of Prolonged Liver Dietary in Pernicious Anemia. Arch. Int. Med., 1932, 50, 124-130.
- 39. Gibson, R. B., and Howard, C. P. Metabolic Studies in Pernicious Anemia. Arch. Int. Med., 1923, 32, 1-16.
- 40. GILROY, E. The Vitamin B Content of Commercial Liver Extracts and Stomach Preparations. Lancet, 1931, 2, 1093-1098.
- 41. Goodall, A. The Treatment of Pernicious Anaemia by Marmite. Lancet, 1932, 2, 781-782.
- 42. GOODALL, A., and SLATER, J. K. Treatment of Disseminated Sclerosis by Liver. Brit. Med. J., 1931, 1, 789-790.
- 43. GRIFFITH, J. P. C., and Scott, J. P. The Employment of Liver for the Anemias of Early Life. Med. J. and Rec., 1928, 128, 121-123.
- 44. GRINKER, R. R. Pernicious Anemia, Achylia Gastrica and Combined Cord Degeneration and Their Relationship. Arch. Int. Med., 1926, 38, 292-302.
- 45. Habershon, S. O. On Idiopathic Anaemia. Lancet, 1863, 1, 518-519, 551-553.
- 46. Hamilton, A. S., and Nixon, C. E. Sensory Changes in the Subacute Combined Degeneration of Pernicious Anemia. Arch. Neurol. and Psychiat., 1921, 6, 1-31.
- 47. HEATH, E. H. Pernicious Anemia Treated with Liver Diet and Liver Extract. J. Amer. Med. Assoc., 1928, 91, 928-932.
- 48. HERRICK, J. B. Nervous Shock and Disease of the Nervous System as a Cause of Pernicious Anemia. J. Amer. Med. Assoc., 1896, 26, 1216-1219.
- 49. HOOPER, C. W., and WHIPPLE, G. H. Blood Regeneration After Simple Anemia. I. Curve of Regeneration Influenced by Dietary Factors. Amer. J. Physiol., 1918, 45, 573-575.
- 50. HULETT, A. G. The Psychological and Medicolegal Aspects of Pernicious Anemia. Med. J. and Rec., 1928, 127, 1-6.
- HUNTER, W. The Nervous and Mental Disorders of Severe Anaemias in Relation to Their Infective Lesions and Blood Changes. Proc. Roy. Soc. Med., 1923, 16, 1-42.
- 52. Hurst, A. F., and Bell, J. R. The Pathogenesis of Subacute Combined Degeneration of the Spinal Cord, with Special Reference to Its Connection with Addison's (Pernicious) Anaemia, Achlorhydria and Intestinal Infection. *Brain*, 1922, 45, 266-281.

- 53. Hurst, A. F. Addison's (Pernicious) Anaemia and Subacute Combined Degeneration of the Spinal Cord. Brit. Med. J., 1924, 1, 93-100.
- 54. Hurst, A. F. The Pathogenesis of Subacute Combined Degeneration of the Spinal Cord with Special Reference to Its Connection with Addison's (Pernicious) Anaemia, Achlorhydria and Intestinal Infection. *Brain*, 1925, 48, 218-232.
- 55. Isaacs, R., Sturgis, C. C., and Smith, M. Treatment of Pernicious Anemia. J. Amer. Med. Assoc., 1928, 91, 1687-1689.
- KESCHNER, M. Nervous and Mental Complications of Pernicious Anemia and the Effect of Liver Therapy Upon Them. Med. J. and Rec., 1931, 133, 598-601.
- 57. Kiely, C. Neurologic and Psychopathic Manifestations of Pernicious Anemia. J. Michigan State Med. Soc., 1932, 31, 272-275.
- 58. Koessler, K. K., Maurer, S., and Richter, O. The Successful Treatment of Severe Pernicious Anemia. *Med. Clin. North Amer.*, 1928, 12, 159-165.
- KOESSLER, K. K., MAURER, S., and LAUGHLIN, R. The Relation of Anemia, Primary and Secondary, to Vitamin A Deficiency. J. Amer. Med. Assoc., 1926, 87, 476-482.
- KOESSLER, K. K., and MAURER, S. Treatment of Pernicious Anemia with a High Caloric Diet, Rich in Vitamins. J. Amer. Med. Assoc., 1927, 89, 768-774.
- 61. Langdon, F. W. Nervous and Mental Manifestations of Pre-pernicious Anemia. J. Amer. Med. Assoc., 1905, 45, 1635-1638.
- 62. Lurie, L. A. Pernicious Anemia with Mental Symptoms. Arch. Neurol. and Psychiat., 1919, 2, 67-109.
- 63. McAlpine, D. Nervous and Mental Aspects of Pernicious Anaemia. Lancet, 1929, 2, 643-647.
- 64. McPeak, E. M., and Neighbors, DeW. Minot-Murphy Diet in Pernicious Anemia: Report of Five Cases. Southern Med. J., 1927, 20, 926-931.
- 65. Macht, D. I. A Study of the Toxin of Pernicious Anemia. Proc. Soc. Exper. Biol. and Med., 1925, 23, 209-210.
- 66. Mason, E. H. Pernicious Anemia. J. Amer. Med. Assoc., 1928, 90, 1527-1529.
- 67. Means, J. H., and Richardson, W. Impressions of Nature of Pernicious Anemia in Light of the Newer Knowledge. J. Amer. Med. Assoc., 1928, 91, 923-925.
- 68. Medical Research Council. An Inquiry into the Results of the Liver Treatment of Pernicious Anaemia. Lancet, 1928, 1, 872-879.
- 69. MELLANBY, E., ET AL. Vitamins: A Survey of Present Knowledge. London: His Majesty's Stationery Office, 1932.
- 70. Mellanby, E. The Experimental Production and Prevention of Degeneration in the Spinal Cord. Brain, 1931, 54, 247-290.
- 71. Mellanby, E. The Relation of Diet to Health and Disease. Brit. Med. J., 1930, 1, 677-681.
- 72. Mellanby, E. Diseases Produced and Prevented by Certain Food Constituents. J. Amer. Med. Assoc., 1931, 96, 325-331.
- 73. Menninger, W. C. Effects of Liver Diet on Nervous System Symptoms in Pernicious Anaemia. Southwestern Med., 1931, 15, 449-453.
- 74. MINOT, G. R., and MURPHY, W. P. Treatment of Pernicious Anemia by a Special Diet. J. Amer. Med. Assoc., 1926, 87, 470-476.
- a Special Diet. J. Amer. Med. Assoc., 1926, 87, 470-476.
 75. MINOT, G. R., and MURPHY, W. P. Treatment of Pernicious (Addi-

- MINOT, G. R., and MURPHY, W. P. Liver Diet in Pernicious Anemia and the Distinction Between Aleukocythemic Myeloid Leukemia and Pernicious Anemia. Med. Clin. North Amer., 1927, 10, 1093-1102.
- 77. MINOT, G. R., and MURPHY, W. P. A Diet Rich in Liver in the Treatment of Pernicious Anemia. J. Amer. Med. Assoc., 1927, 89, 759-766.
- 78. Moschcowitz, E. The Relation of Achlorhydria to Pernicious Anemia.

 Arch. Int. Med., 1931, 48, 171-186.
- 79. Murphy, W. P. Observations on the Treatment of Anemia. Surg., Gynecol. and Obstet., 1930, 50, 246-250.
- 80. Needles, W. Neurologic Complications of Pernicious Anemia. Arch. Neurol. and Psychiat., 1931, 26, 346-358.
- 81. ORDWAY, T., and GORHAM, L. W. The Treatment of Pernicious Anemia with Liver and Liver Extract. J. Amer. Med. Assoc., 1928, 91, 925-928.
- 82. Pepper, O. H. P. A Medical Clinic. J. Iowa State Med. Soc., 1930, 20, 495-498.
- 83. Perkins, J. N. Combined Sclerosis Associated with Pernicious Anemia. U. S. Vet. Bur. Med. Bull., 1930, 6, 367-370.
- 84. Pfeffer, T. J. Pernicious Anemia and Subacute Combined Degeneration of the Spinal Cord. J. Iowa State Med. Soc., 1932, 22, 114-118.
- 85. PFEIFFER, J. A. F. The Neuropathological Findings in a Case of Pernicious Anemia with Psychical Implication. J. Nerv. and Ment. Dis., 1915, 42, 75-93.
- 86. Pickett, W. Mental Symptoms Associated with Pernicious Anemia. Amer. J. Med. Sci., 1904, 127, 1032-1036.
- 87. PINEY, A. Mental Changes Associated with Pernicious Anaemia. J. Neurol. and Psychopath., 1932, 13, 127-132.
- 88. Pollard, J. B. Pernicious Anemia. U. S. Naval Med. Bull., 1925, 22, 649-655.
- 89. Reed, A. C., and Wyckoff, H. A. The Common Picture of Sprue, Pernicious Anemia, and Combined Degeneration. Amer. J. Trop. Med., 1926, 6, 221-237.
- REESE, H. H., and BEIGLER, S. K. Subacute Combined Degeneration of the Spinal Cord and Pernicious Anemia. Amer. J. Med. Sci., 1926, 171, 194-202.
- 91. RICHARDS, G. G., and DAINES, L. L. Recent Observations in Pernicious Anemia. Ann. Clin. Med., 1926-1927, 5, 966-971.
- 92. RICHARDSON, W. Pernicious Anaemia. New Eng. J. Med., 1929, 200, 540-545.
- 93. Riggs, C. E. Some Nervous Symptoms of Pernicious Anemia. J. Amer. Med. Assoc., 1913, 61, 481-484.
- 94. Rubenstone, A. I. Pernicious Anemia. Med. J. and Rec., 1931, 133, 594-597.
- 95. Scatliff, H. K. Pernicious Anemia and the Emotions. Illinois Med. J., 1931, 59, 394-396.
- 96. Science News. Vitamin B₂ and Pernicious Anemia. Science, supplement, 1933, 77, 7-8.
- 97. SHARP, E. A. An Antianemic Factor in Desiccated Stomach. J. Amer. Med. Assoc., 1929, 93, 749.
- 98. SIMMONDS, N., BECKER, J. E., and McCollum, E. V. The Relation of Vitamin E to Iron Assimilation. J. Amer. Med. Assoc., 1927, 88, 1047-1050.

- 99. SMITH, L. H. Mental and Neurologic Changes in Pernicious Anemia.
- Arch. Neurol. and Psychiat., 1929, 22, 551-557.

 100. SMITHBURN, K. C., and ZERFAS, L. G. The Neural Symptoms and Signs in Pernicious Anemia. Arch. Neurol. and Psychiat., 1931, 25, 1100-
- The Prevention of Spinal Cord Degeneration in Pernicious 101. STARR, P. Anemia. J. Amer. Med. Assoc., 1931, 96, 1219-1221.
- 102. STURGIS, C. C., ISAACS, R., and RIDDLE, M. C. The Treatment of Pernicious Anemia by Liver Feeding. Surg., Gynecol. and Obstet., 1930, **50**, 234–243.
- 103. STURGIS, C. C., and ISAACS, R. Desiccated Stomach in the Treatment of Pernicious Anemia. J. Amer. Med. Assoc., 1929, 93, 747-749.
- 104. STURGIS, C. C., and ISAACS, R. Clinical and Experimental Observations on the Treatment of Pernicious Anemia with Desiccated Stomach and
- with Liver Extract. Ann. Int. Med., 1931, 5, 131-158.

 105. Sure, B., Kik, M. C., and Smith, M. E. Hematopoietic Function in Avitaminosis. VI. Vitamin G Deficiency. Proc. Soc. Exper. Biol. and Med., 1930-1931, 28, 498-499.
- 106. Ungley, C. C., and Suzman, M. M. Subacute Combined Degeneration of the Cord: Symptomatology and Effects of Liver Therapy. Brain, 1929, **52**, 271–294.
- 107. Ungley, C. C. Effect of Brain Diet in Subacute Combined Degeneration of the Cord. Lancet, 1932, 1, 227-230.
 108. Ungley, C. C. Liver Treatment in Subacute Combined Degeneration of
- the Cord. Lancet, 1929, 2, 794.
- 109. VAN WART, R. M. The Nervous Symptoms Accompanying Pernicious Anemia. Med. News, 1905, 86, 56-59.
- 110. VAUGHAN, J. Investigation of a Series of Cases of Secondary Anaemia. Lancet, 1928, 1, 1063-1066.
- 111. VAUGHAN, J. M. Critical Review: the Liver Treatment of Anaemias. Quart. J. Med., 1930, 23, 213-232.
- 112. VAUGHAN, J. Discussion on the Value of Liver in Treatment. Proc. Royal Soc. Med., 1931, 24, 929-935.
- 113. WARFIELD, L. M. Some Neurological Manifestations of Pernicious Anemia. Wisconsin Med. J., 1922, 21, 54-57.
- 114. Weisenburg, T. H. Neurological Symptoms Occurring in Pernicious Anemia Especially Antedating the Appearance of the Blood Picture. Med. Rec., 1921, 99, 942.
- 115. DE WESSELOW, O. L. V., and BAMFORTH, J. The Blood and Plasma Volumes in Pernicious Anemia. Lancet, 1928, 1, 1066-1068.
- 116. West, R. Pernicious Anemia as a Deficiency Disease. Ann. Int. Med., 1929, 3, 132-136.
- 117. Whipple, G. H. Experimental Anemias, Diet Factors and Related Pathologic Changes of Human Anemias. J. Amer. Med. Assoc., 1928, **91**, 863–867.
- 118. WILKINSON, J. F. Treatment of Pernicious Anaemia with Hog's Stomach. Brit. Med. J., 1931, 1, 85-91.
- 119. WILKINSON, J. F. Discussion on the Value of Liver in Treatment. Proc. Royal Soc. Med., 1931, 24, 948-951.
- 120. WINKELMAN, N. W., and ECKEL, J. L. Pernicious Anemia: Difficulties in Diagnosis from a Neurological Standpoint. New York State J. Med., 1929, 29, 313-318.
- 121. WOLTMAN, H. W. Brain Changes Associated with Pernicious Anemia. Arch. Int. Med., 1918, 21, 791-843.

- 122. WOLTMAN, H. W. Neurologic Aspects of the Early Diagnosis of Pernicious Anemia. Ann. Clin. Med., 1922-1923, 1, 159-160.
- 123. WOLTMAN, H. W. The Nervous Symptoms in Pernicious Anemia: An Analysis of One Hundred Fifty Cases. Amer. J. Med. Sci., 1919, 157,
- 124. WOLTMAN, H. W. The Mental Changes Associated with Pernicious Anemia. Amer. J. Psychiat., 1924, 3, 435-449.
- 125. Young, R. H. Neurologic Features of Pernicious Anemia. J. Amer. Med. Assoc., 1932, 99, 612-614.
- 126. Young, A. W. The Neurological Complications of Pernicious Anemia and the Effect of Liver Therapy. Canadian Med. Assoc. J., 1932, 26, 590-593.

Pellagra

- 127. AYKROYD, W. R. The Etiology of Pellagra. Brit. Med. J., 1930, 1, 647-648.
- 128. BAILLARGER, M. The Pellagra. J. Psychol. Med. and Mental Path., 1848, 1, 460–466.
- 129. Bellamy, R. H. Pellagra. J. Amer. Med. Assoc., 1908, 51, 397-399.
- 130. BIGLAND, A. D. Pellagra in the British Islands. Lancet, 1923, 2, 1295-
- 131. Bliss, S. Considerations Leading to the View that Pellagra is an Iron-Deficiency Disease. Science, 1930, 72, 577-578.
- 132. Carley, P. S. The Use of Dried Brewers Yeast in the Treatment and Prevention of Pellagra. New Orleans Med. and Surg. J., 1930, 82, 740-744.
- 133. Cooper, T. C. Pellagrous Insanity. Amer. J. Psychiat., 1928, 7, 945-952. 134. Davis, T. K. A Case of Pellagra with Neurologic Manifestations. Arch. Neurol. and Psychiat., 1931, 26, 438-439.
- 135. Enright, J. I. The Pellagra Outbreak in Egypt. Lancet, 1920, 1, 998-1003.
- 136. FARNELL, F. J., and YACOVLEV, P. J. Pellagra and Beri-beri. Ann. Clin. Med., 1926, 4, 541-551.
- 137. FAUST-NEWTON, C. Dietary Factor in Treatment and Prevention of Pellagra, with Particular Reference to Yeast. South. Med. J., 1926, **19**, 168–175.
- 138. Frazer, T. Mental and Nervous Manifestations of Pellagra. Med. Rec., 1914, 86, 65-67.
- 139. GARDNER, W. E. Pellagra as Related to Insanity. Kentucky Med. J., 1912-1913, 11, 353-357.
- 140. GOLDBERGER, J. Pellagra: Causation and a Method of Prevention. J. Amer. Med. Assoc., 1916, 66, 471-476.
- 141. GOLDBERGER, J. The Relation of Diet to Pellagra. J. Amer. Med. Assoc., 1922, 78, 1676–1680.
- 142. Goldberger, J. Pellagra: Its Nature and Prevention. U. S. Pub. Health Rep., 1927, 42, 2193-2200.
- 143. Goldberger, J. Pellagra. J. Amer. Dietetic Assoc., 1929, 4, 221-227.
- 144. GOLDBERGER, J., and TANNER, W. F. Amino-acid Deficiency Probably the Primary Etiological Factor in Pellagra. U. S. Pub. Health Rep., 1922, **37**, 462–486.
- 145. GOLDBERGER, J., and TANNER, W. F. A Study of the Treatment and Prevention of Pellagra. U. S. Pub. Health Rep., 1924, 39, 87-107.

- 146. Goldberger, J., and Tanner, W. F. A Study of the Pellagra-Preventive Action of Dried Beans, Casein, Dried Milk, and Brewer's Yeast, with a Consideration of the Essential Preventive Factors Involved. U. S. Pub. Health Rep., 1925, 40, 54-80.
- 147. GOLDBERGER, J., WARING, C. H., and WILLETS, D. G. The Treatment and Prevention of Pellagra. U. S. Pub. Health Rep., 1914, 29, 2821-2825.
- 148. Goldberger, J., and Wheeler, G. A. Experimental Pellagra in the Human Subject Brought About by a Restricted Diet. U. S. Pub. Health Rep., 1915, 30, 3336-3339.
- 149. Goldberger, J., and Wheeler, G. A. A Study of the Blacktongue-Preventive Action of 16 Foodstuffs, with Special Reference to the Identity of Blacktongue of Dogs and Pellagra of Man. U. S. Pub. Health Rep., 1928, 43, 1385-1454.
- 150. Goldberger, J., Wheeler, G. A., and Sydenstricker, E. A Study of the Diet of Non-Pellagrous and of Pellagrous Households. J. Amer. Med. Assoc., 1918, 71, 944-949.
- 151. Green, E. M. Psychoses Accompanying Pellagra. J. Amer. Med. Assoc., 1912, 58, 1779.
- 152. GREER, A. E. Pellagra-Like Skin Lesions Due to the Ketogenic Diet. J. Amer. Med. Assoc., 1930, 95, 863.
- 153. GRIMM, R. M. Pellagra. U. S. Pub. Health Rep., 1913, 28, 427-450, 491-513.
- 154. Guha, B. C. Vitamin B2 and Pellagra. Brit. Med. J., 1931, 2, 53-54.
- 155. GUTHRIE, R. H. Pellagra-Like Skin Lesions Appearing in the Course of a Ketogenic Diet. J. Amer. Med. Assoc., 1930, 95, 1912-1913.
- 156. HARRIS, H. F. Ankylostomiasis in an Individual Presenting All of the Typical Symptoms of Pellagra. Amer. Med., 1902, 4, 99-100.
- 157. HARRIS, H. F. The Diagnosis of Pellagra. Amer. J. Med. Sci., 1911, 141, 715-724.
- 158. HARRIS, S. The Food Factor in Pellagra. Internat. Clin., 1924, 4, 34th series, 21-31.
- 159. HAYNES, B. Pellagra from the Viewpoint of the Patient. Internat. Clin., 1926, 4, 36th series, 76-80.
- HINDHEDE, M. Protein and Pellagra. J. Amer. Med. Assoc., 1923, 80, 1685-1689.
- 161. Hoag, D. E. Pellagra: Observations on Some of Its Nervous Manifestations. J. Amer. Med. Assoc., 1912, 59, 1445-1447.
- 162. Hyde, J. N. Pellagra and Some of Its Problems. Amer. J. Med. Sci., 1910, 139, 1-26.
- 163. Illinois Pellagra Commission. Pellagra in Illinois. Arch. Int. Med., 1912, 10, 123-168, 219-249.
- 164. Jobling, J. W., and Arnold, L. Observation and Reflections on the Etiology of Pellagra. J. Amer. Med. Assoc., 1923, 80, 365-368.
- 165. Johnson, F. Results of Some Studies Dealing with Diets in Pellagra. Southern Med. and Surg., 1932, 94, 130-136.
- 166. Koch, M. L., and Voegtlin, C. I. Chemical Changes in the Central Nervous System as a Result of Restricted Vegetable Diet. II. Chemical Changes in the Central Nervous System in Pellagra. U. S. Hygienic Lab. Bull., No. 103, 1916. Pp. 129.
- 167. Langworthy, O. R. Lesions of the Central Nervous System Characteristic of Pellagra. Brain, 1931, 54, 291-302.
- 168. LAVINDER, C. H. Notes on the Prognosis and Treatment of Pellagra. U. S. Pub. Health Rep., 1909, 24, 1315-1321.

- 169. LEADER, V. R. Some Factors Involved in the Experimental Production of Pellagra in Rats. Biochem. J., 1930, 24, 1172-1180.
- 170. Lorenz, W. F. The Treatment of Pellagra. U. S. Pub. Health Rep., 1914, 29, 2357-2360.
- 171. LORENZ, W. F. Mental Manifestations of Pellagra. U. S. Pub. Health Rep., 1916, 31, 221-246.
- 172. Lowery, J. R. Pellagra. Med. Rec., 1914, 86, 378-379.
- 173. Lustberg, S. R., and Birchett, J. A. K. The Breast Fed Pellagrin: Relation to the Avitaminoses. Arch. Ped., 1922, 39, 255-258.
- 174. McCollum, E. V., and Simmonds, N. A Biological Analysis of Pellagra-Producing Diets. J. Biol. Chem., 1917, 32, 347-368.
- 175. MACDONALD, J. B. Pellagra and Its Symptoms; the Importance of Mouth and Gastrointestinal Lesions. Boston Med. and Surg. J., 1914, 171, 485-489.
- 176. MacNeal, W. J. The Alleged Production of Pellagra by an Unbalanced Diet. J. Amer. Med. Assoc., 1916, 66, 975-977.
- 177. MACNEAL, W. J. Pellagra. Amer. J. Med. Sci., 1921, 161, 469-501.
- 178. Morel, B. A. On the Degeneracy of the Human Race. J. Psychol. Med. and Mental Path., 1857, 10, 159-208.
- 179. NESBITT, C. T. Sanitation and the Control of Pellagra. J. Amer. Med. Assoc., 1916, 66, 647-648.
- 180. O'LEARY, P. A. Secondary Types of Pellagra. Med. Clin. North Amer., 1926, 10, 647-658.
- PIERCE, L. B. Pellagra. Report of a Case. Amer. J. Psychiat., 1924, 4, 237-243.
- 182. Pound, J. H. Pellagra with Psychoses. J. Florida Med. Assoc., 1928, 15, 299-302.
- 183. RANDOLPH, J. H. Notes on Pellagra and Pellagrins. Arch. Int. Med., 1908-1909, 2, 553-568.
- 184. Ridlon, J. R. Pellagra. The Value of the Dietary Treatment of the Disease. U. S. Pub. Health Rep., 1916, 31, 1979-1999.
- 185. Roberts, S. R. Pellagra of Today. Internat. Clin., 1929, 1, 39th series, 65-76.
- 186. Roberts, S. R. Acute Pellagra Associated with a Manic-Depressive Psychosis Developing After a Five-Year Pre-Pellagrous Period. Med. Clin. North Amer., 1929, 12, 1421-1427.
- 187. Sabry, I. On the Chemical Nature of Pellagra Toxin. Lancet, 1931, 2, 1020-1022.
- 188. SANDELS, M. R., and GRADY, E. Dietary Practices in Relation to the Incidence of Pellagra. Arch. Int. Med., 1932, 50, 362-372.
- 189. SANDWITH, F. N. Pellagra in Egypt. Brit. Med. J., 1898, 2, 881.
- 190. SEARCY, G. H. An Epidemic of Acute Pellagra. J. Amer. Med. Assoc., 1907, 49, 37-38.
- 191. Shattuck, G. C. Factors Apparently Influencing the Development of Pellagra in Massachusetts. *Boston Med. and Surg. J.*, 1923, 188, 889-891.
- 192. SINGER, H. D. Mental and Nervous Disorders Associated with Pellagra. Arch. Int. Med., 1915, 15, 121-146.
- 193. SINGER, H. D., and POLLOCK, L. J. The Histopathology of the Nervous System in Pellagra. Arch. Int. Med., 1913, 11, 565-589.
- 194. SMITH, J. H. The Influence of Solar Rays on Metabolism. With Special Reference to Sulphur and to Pellagra in Southern United States. Arch. Int. Med., 1931, 48, 907-1063.

- 195. Spies, T. D. Pellagra: Etiology; Response to a Deficient Diet. Southern Med. and Surg., 1932, 94, 128-129.
- 196. THATCHER, H. S. Pellagra. Arch. Path., 1931, 12, 970-982.
- 197. THAYER, W. S. Note on Pellagra in Maryland. Bull. Johns Hopkins Hosp., 1909, 20, 193-200.
- 198. Thurlow, A. A. A Clinical Observation on Psychosis Accompanying Pellagra. Illinois Med. J., 1914, 26, 342-343.
- 199. Tucker, B. R. Pellagra in Its Relation to Neurology and Psychiatry. Amer. J. Med. Sci., 1912, 143, 332-339.
- 200. UNDERHILL, F. P. Clinical Aspects of Vitamin G Deficiency. J. Amer. Med. Assoc., 1932, 99, 120-124.
- 201. VEDDER, E. B. Dietary Deficiency as the Etiological Factor in Pellagra. Arch. Int. Med., 1916, 18, 137-172.
- 202. VISWALINGAM, A. Some Further Observations on the Ætiology of "Pellagra." J. Trop. Med. and Hygiene, 1920, 23, 46-47.
- 203. Voegtlin, C. Recent Work on Pellagra. U. S. Pub. Health Rep., 1920, 35, 1435-1452.
- 204. WALKER, N. P. Yeast and Pellagra. J. Med. Assoc. of Georgia, 1929, 18, 379-380.
- 205. WARNOCK and DUDGEON. Twenty-fifth Annual Report of Government Hospital for the Insane at Abbasayi, 1920. (P. 203 in McCarrison, R., Studies in Deficiency Disease. London: Frowde, Hodder and Stoughton, 1921. Pp. 270.)
- WATSON, J. J. Pellagra. New York Med. J., 1909, 89, 936-941.
- 207. WHEELER, G. A., and SEBRELL, W. H. The Control of Pellagra. Amer. Med. Assoc., 1932, 99, 95-98.
- 208. Wholey, C. C. Nervous and Mental Symptoms in a Case of Pellagra. Pennsylvania Med. J., 1923, 26, 318-321.
- 209. WILSON, W. H. The Diet Factor in Pellagra. J. Hygiene, 1921, 20, 1-59. 210. WILSON, W. H. Note on the Etiology of Pellagra. Brit. Med. J., 1930, 1, 101-103.
- 211. Wood, E. J. A Treatise on Pellagra. New York: D. Appleton & Co., 1912.
- 212. Wood, E. J. Vitamin Solution of the Pellagra Problem. J. Amer. Med. Assoc., 1916, 66, 1447-1448.
- 213. Wood, E. J. The Etiology of Pellagra; a Consideration of Vitamin Deficiency. Amer. J. Med. Sci., 1916, 152, 813-823.
- 214. Wood, E. J. Pellagra. Edinburgh Med. J., 1920, 25, 363-374.

Sprue

- 215. ASHFORD, B. K. The Etiology of Sprue. Amer. J. Med. Sci., 1917, 154, 157-176.
- 216. ASHFORD, B. K. Observations on the Conception that Sprue is a Mycosis Superimposed Upon a State of Deficiency in Certain Essential Food Elements. Amer. J. Trop. Med., 1922, 2, 139-150.
- 217. ASHFORD, B. K. A Clinical Investigation of Tropical Sprue. Amer. J. Med. Sci., 1923, 165, 157-173.
- 218. BAUMGARTNER, E. A., and SMITH, G. D. Pernicious Anemia and Tropical Sprue. Arch. Int. Med., 1927, 40, 203-215.
- 219. BLOOMFIELD, A. L., and WYCKOFF, H. A. Remission in Sprue Following High Liver Diet: Case Report. California and Western Med., 1927, 27, 659.

- 220. ELDERS, C. Tropical Sprue and Pernicious Anaemia. Lancet, 1925, 1, 75-77.
- 221. Fontaine, B. W. A Case of Tropical Sprue Endemic in Tennessee. Diagnosis and Treatment. *Med. Clin. North Amer.*, 1929, 12, 1223-1243.
- 222. Manson-Bahr, P., and Willoughby, H. Studies on Sprue with Special Reference to Treatment. Quart. J. Med., 1930, 23, 411-442.
- 223. MINOT, G. R., MURPHY, W. P., and STETSON, R. P. The Response of the Reticulocytes to Liver Therapy: Particularly in Pernicious Anemia. Amer. J. Med. Sci., 1928, 175, 581-599.
- 224. Musser, J. H. Clinical Manifestations of Sprue and Relation of the Disease to Pernicious Anemia. Med. Clin. North Amer., 1926, 9, 895-908.
- 225. Nye, R. N., Zerfas, L. G., and Cornwell, M. A. The Presence and Importance of Yeastlike Fungi in the Gastrointestinal Tract in Pernicious Anemia, in Other Diseases and in Normal Individuals. Amer. J. Med. Sci., 1928, 175, 153-174.
- 226. Wood, E. J. The Occurrence of Sprue in the United States. Amer. J. Med. Sci., 1915, 150, 692-699.
- 227. Wood, E. J. Pernicious Anemia in Its Relationship to Sprue. Amer. J. Med. Sci., 1925, 169, 28-38.
 Also, see No. 89.

Acrodynia

- 228. Bilderback, J. B. A Group of Cases of Unknown Etiology and Diagnosis. Northwest Med., 1920, 19, 263-265.
- 229. BILDERBACK, J. B. Acrodynia. J. Amer. Med. Assoc., 1925, 84, 495-498.
- 230. Brown, A., Courtney, A. M., and MacLachlan, I. F. A Clinical and Metabolic Study of Acrodynia. Arch. Ped., 1921, 38, 609-628.
- 231. BUTLER, J. Erythredema. Arch. Dermat. and Syph., 1925, 11, 166-182.
- 232. Byfield, A. H. A Polyneuritic Syndrome Resembling Pellagra-Acrodynia (?) Seen in Very Young Children. Amer. J. Dis. Child., 1920, 20, 347-365.
- 233. CARTIN, H. J. Acrodynia. Pennsylvania Med. J., 1921, 24, 287-289.
- 234. CRAIG, R. A. Acrodynia. Arch. Ped., 1927, 44, 581-585.
- 235. EMERSON, P. W. A Case of Acrodynia. J. Amer. Med. Assoc., 1921, 77, 285-286.
- 236. FIELD, M. C. A Case of Erythroedema. Arch. Ped., 1922, 39, 116-121.
- 237. FOERSTER, H. R. Erythredema Polyneuritis. Arch. Dermat. and Syph., 1925, 12, 17-32.
- 238. GIFFEN, S. D. Acrodynia. J. Michigan State Med. Soc., 1924, 23, 8-14.
- 239. Goodman, H., and Burr, M. Juvenile Acrodynia. Arch. Dermat. and Syph., 1931, 23, 901-907.
- 240. Guralnick, R. Two Cases of Acrodynia. New England J. Med., 1930, 202, 476-477.
- 241. Helmick, A. G. Symptomatology of Acrodynia as a Basis for a New Line of Investigation as to Its Etiology. *Arch. Ped.*, 1927, 44, 405-410.
- 242. HELMICK, A. G. Acrodynia. Ohio State Med. J., 1928, 24, 123-126.
- 243. Kernohan, J. W., and Kennedy, R. L. J. Acrodynia (so-called). Amer. J. Dis. Child., 1928, 36, 341-351.
- 244. Kugelmass, I. N. Nutritional Diseases. Internat. Clin., 1932, 2, 42nd series, 255-264.

- 245. McClendon, S. J. Yeast and Irradiated Ergosterol in the Treatment of Acrodynia. J. Amer. Med. Assoc., 1929, 93, 455.
- 246. McNeal, M. D. A Report of Five Cases of So-Called Acrodynia. Minnesota Med., 1922, 5, 153-156.
- 247. MILLER, M. K. Polyneuritic Syndrome in Young Children. J. Indiana State Med. Assoc., 1921, 14, 144-145.
- 248. NESBIT, H. T. Acrodynia: Its Etiology. Arch. Ped., 1932; 49, 135-140.
- 249. Parsons, L. Exhibition of Cases and Specimens ("Pink Disease"). Lancet, 1923, 1, 490.
- 250. PARSONS, L. G. Pink Disease. Practitioner, 1930, 125, 146-155.
- 251. Perlman, H. H. Report of a Case of Acrodynia with Complete Recovery. Med. J. and Rec., 1929, 130, 370-375.
- 252. Powell, W. Acrodynia. With Report of Cases. Boston Med. and Surg. J., 1926, 194, 980-984.
- 253. RODDA, F. C. Acrodynia. Amer. J. Dis. Child., 1925, 30, 224-231.
- 254. Sweet, G. B. Acrodynia. Arch. Ped., 1925, 42, 543-549.
- 255. Thursfield, H. Dermato-Polyneuritis (Acrodynia: Erythroedema). Brit. J. Dis. Child., 1922, 19, 27-31.
- 256. VIPOND, A. E. Acrodynia and Its Probable Causation. Arch. Ped., 1922, 39, 699-704.
- 257. WATTS, A. F. Acrodynia. J. Iowa State Med. Soc., 1931, 21, 291-295.
- 258. Weber, F. P. Case of Erythroedema (the "pink disease"); and the Question of Acrodynia ("epidemic erythema"). Brit. J. Child. Dis., 1922, 19, 17-27.
- 259. Weston, W. Acrodynia in the United States. Southern Med. J., 1926, 19, 665-669.
- 260. Weston, W. The Food Elements as Factors in Preventive Medicine. J. South Carolina Med. Assoc., 1927, 23, 489-495.
- 261. WHITE, C. J. Acrodynia in Adults. J. Amer. Med. Assoc., 1926, 87, 1092-1095.
- 262. Wood, E. J. Acrodynia: Its Place in Medicine and Its Relation to Pellagra. Amer. J. Trop. Med., 1921, 1, 291-310.
- 263. Wood, E. J. Is Acrodynia a New Disease? J. Amer. Med. Assoc., 1925, 85, 1419-1420.
- 264. WYCKOFF, C. W. Juvenile Acrodynia. Amer. J. Dis. Child., 1929, 37, 88-97.
- 265. WYLLIE, W. G., and STERN, R. O. Pink Disease: Its Morbid Anatomy, with Note on Treatment. Arch. Dis. in Childhood, 1931, 6, 137-156.
- 266. Zahorsky, J. The Semeiology of the Pink Disease (Erythredema, Acrodynia). J. Amer. Med. Assoc., 1922, 79, 1975-1979.
- 267. ZAHORSKY, J. Pink Disease Treated with Yeast. Amer. J. Dis. Child., 1929, 37, 449.

Migraine

- 268. Allan, W. The Relation of Occupation to Migraine. J. Nerv. and Ment. Dis., 1927, 66, 131-132.
- 269. ALLAN, W. The Neuropathic Taint in Migraine. Arch. Neurol. and Psychiat., 1927, 18, 587-590.
- 270. ALLAN, W. The Inheritance of Migraine. Arch. Int. Med., 1928, 42, 590-599.
- 271. ALLAN, W. Status Hemicranicus and the Frequency of Migraine Attacks. J. Nerv. and Ment. Dis., 1928, 68, 591-593.

273. Ball, F. E. Migraine—Its Treatment with Peptone and Its Familial Relation to Sensitization Diseases. Amer. J. Med. Sci., 1927, 173, 781-788.

274. BALYEAT, R. M., and BRITTAIN, F. L. Allergic Migraine. Amer. J. Med. Sci., 1930, 180, 212-221.

275. BALYEAT, R. M., and RINKEL, H. J. Allergic Migraine in Children. Amer. J. Dis. Child., 1931, 42, 1126-1133.

276. BALYEAT, R. M., and RINKEL, H. J. Further Studies in Allergic Migraine: Based on a Series of Two Hundred and Two Consecutive Cases. Ann. Int. Med., 1931, 5, 713-728.

277. BARBORKA, C. J. The Ketogenic Diet and Its Use. Med. Clin. North Amer., 1929, 12, 1649-1653.

278. BARBORKA, C. J. Migraine. Results of Treatment by Ketogenic Diet in Fifty Cases. J. Amer. Med. Assoc., 1930, 95, 1825-1828.

279. BEECHER, W. L. Migraine or Sick Headache. Illinois Med. J., 1929, 55, 123-125.

280. BIGLAND, A. D. Treatment of Migraine by Calcium Lactate. Brit. Med. J., 1923, 2, 1133-1135.

281. Bramwell, E. Migraine. Brit. Med. J., 1926, 2, 765-769.

282. Brown, R. C. The Protein of Foodstuffs as a Factor in the Cause of Headache. Wisconsin Med. J., 1920, 19, 337-346.

283. Brown, T. R. Rôle of Diet in Etiology and Treatment of Migraine and Other Types of Headache. J. Amer. Med. Assoc., 1921, 77, 1396-1400.

284. Brown, T. R. Thoughts on the Modern Methods of Diagnosis and Treatment in Digestive Diseases. *Internat. Clin.*, 1923, 1, 33rd series, 27-49.

285. Brown, T. R. Referred Symptoms in Gastro-Intestinal Diseases and Digestive Symptoms in Other Diseases. *Internat. Clin.*, 1932, 2, 42nd series, 60-94.

286. Buchanan, J. A. The Mendelianism of Migraine. *Med. Rec.*, 1920, 98, 807-808.

287. Curtis-Brown, R. A Protein Poison Theory. Brit. Med. J., 1925, 1, 155-156.

288. DeGowin, E. L. Allergic Migraine. A Review of Sixty Cases. J. Allergy, 1931-1932, 3, 557-566.

289. DIAMOND, J. S. Liver Dysfunction in Migrain. Amer. J. Med. Sci., 1927, 174, 695-702.

290. EASTLAKE, C. Allergic Epilepsy. Colorado Med., 1925, 22, 353-355.

291. ELY, F. A. The Migraine-Epilepsy Syndrome. Arch. Neurol. and Psychiat., 1930, 24, 943-949.

292. EYERMANN, C. H. Allergic Headache. J. Allergy, 1930-1931, 2, 106-112.

293. Foley, F. E. B. Clinical Uses of Salt Solution in Conditions of Increased Intracranial Tension. Surg., Gynecol. and Obst., 1921, 33, 126.

294. Gordon, A. H. Some Aspects of Migraine. Internat. Clin., 1924, 1, 34th series, 120-131.

295. Gould, G. M. The History and Etiology of "Migraine." J. Amer. Med. Assoc., 1904, 42, 168-172, 239-244.

296. Gowers, W. R. Borderland of Epilepsy: III. Migraine. Brit. Med. J., 1906, 2, 1617-1622.

297. GRIMES, E. The Migraine Instability. Med. J. and Rec., 1931, 134, 417-422.

298. HARTSOCK, C. L. Migraine. J. Amer. Med. Assoc., 1927, 89, 1489-1492.

- 299. HARTUNG, E. F. Endocrine Factors in Migraine. Med. J. and Rec., 1930, 132, 497-500.
- 300. Hubbell, A. A. Relation of So-Called Ophthalmic Migraine to Epilepsy. J. Amer. Med. Assoc., 1908, 51, 480-482.
- 301. Hughson, W. A Method for the Administration of Sodium Chlorid for Headaches. J. Amer. Med. Assoc., 1921, 77, 1859-1860.
- 302. Hunt, J. R. A Contribution to the Paralytic and Other Persistent Sequelae of Migraine. Amer. J. Med. Sci., 1915, 150, 313-330.
- 303. Hurst, A. F. Migraine. Lancet, 1924, 207, 1-6.
- 304. Kennedy, F. Migraine. Yale J. Biol. and Med., 1929-1930, 2, 61-62.
- 305. Kennedy, F. Migraine: A Localized Intracranial Edema. Internat. Clin., 1931, 3, 41st series, 200-204.
- 306. Kugelmass, I. N. Allergic Diseases. Internat. Clin., 1932, 2, 42nd series, 264-268.
- 307. VAN LEEUWEN, W. S., and ZEYDNER. On the Occurrence of a Toxic Substance in the Blood in Cases of Bronchial Asthma, Urticaria, Epilepsy, and Migraine. Brit. J. Exper. Path., 1922, 3, 282-286.
- 308. McClure, C. W., and Huntsinger, M. E. Observations on Migraine. Boston Med. and Surg. J., 1927, 196, 270-273.
- 309. McClure, C. W., and Huntsinger, M. E. Paroxysmal Headache. II. Observations on the Etiology, Symptomatology, and Treatment of the Migrainous State. New England J. Med., 1928, 199, 1312-1317.
- 310. McMullen, W. H. Migraine from the Ophthalmic Standpoint. Brit. Med. J., 1926, 2, 769-771.
- 311. MILLER, J. L., and RAULSTON, B. O. Treatment of Migraine with Peptone. J. Amer. Med. Assoc., 1923, 80, 1894-1896.
- 312. Minot, G. R. The Rôle of a Low Carbohydrate Diet in the Treatment of Migraine and Headache. Med. Clin. North Amer., 1923, 7, 715-728.
- 313. Moersch, F. P. Psychic Manifestations in Migraine. Amer. J. Psychiat., 1924, 3, 697-716.
- 314. Moloney, J. C. The Etiology of Migraine. Arch. Neurol. and Psychiat., 1928, 19, 684-688.
- 315. Pollock, L. W., and Barborka, C. J. Abdominal Migraine. Med. Clin. North Amer., 1928, 11, 1665-1667.
- 316. RICHET, C. Food Anaphylaxis. J. Allergy, 1930-1931, 2, 76-84.
- 317. RILEY, W. H. Headaches. Battle Creek, Michigan: Good Health Pub. Co., 1916.
- 318. RINKEL, H. J., and BALYEAT, R. M. Pathology and Symptomatology of Headaches Due to Specific Sensitization. J. Amer. Med. Assoc., 1932, 99, 806-811.
- 319. Rowe, A. H. Allergy in the Etiology of Disease. J. Lab. and Clin. Med., 1927-1928, 13, 31-40.
- 320. Rowe, A. H. Food Allergy. Its Manifestation, Diagnosis and Treatment. J. Amer. Med. Assoc., 1928, 91, 1623-1631.
- 321. Rowe, A. H. Allergic Toxemia and Migraine Due to Food Allergy. California and Western Med., 1930, 33, 785-792.
- 322. Rowe, A. H. Allergic Migraine. J. Amer. Med. Assoc., 1932, 99, 912-
- 323. RUPERT, M. P. S., and WILSON, E. E. A Study of Migraine. Amer. J. Med. Sci., 1919, 157, 361-366.
- 324. Schnabel, T. G. An Experience with a Ketogenic Dietary in Migraine. Ann. Int. Med., 1928, 2, 341-347.
- 325. Sherwood-Dunn, B. The Cause of Chronic Diseases, and Their Treatment by Entero-Antigens. Internat. Clin., 1923, 4, 33rd series, 60-76.

- 326. STEVENS, N. C. Endocrine and Fatigue Headaches. New England J. Med., 1929, 201, 801-805.
- 327. THOMAS, J. J. Migraine and Hemianopsia. J. Nerv. and Ment. Dis., 1907, 34, 153–171.
- 328. TILESTON, W. Migraine in Childhood. Amer. J. Dis. Child., 1918, 16, 312-317.
- 329. VAUGHAN, W. T. Diseases Associated with Protein Sensitization. Virginia Med. Month., 1922, 49, 316-322.
- 330. VAUGHAN, W. T. Allergic Migraine. J. Amer. Med. Assoc., 1927, 88, 1383–1386.

Epilepsy

- 331. BARBORKA, C. J. Ketogenic Diet Treatment of Epilepsy in Adults. J. Amer. Med. Assoc., 1928, 91, 73-78.
- 332. BARBORKA, C. J. Ketogenic Diet in Epilepsy. Mod. Hosp., 1928, 30, 144-146.
- 333. BARBORKA, C. J. The Ketogenic Diet and Its Use. Med. Clin. North Amer., 1929, 12, 1639-1649.
- 334. BARBORKA, C. J. Epilepsy in Adults. Arch. Neurol. and Psychiat., 1930, **23**, 904–914.
- 335. Barnes, F. M. Epileptic Mental Disorders. Med. Clin. North Amer., 1927, 11, 425–438.
- 336. Boyd, W. A. Epilepsy: Differential Diagnosis and Treatment. U. S. Vet. Bur. Med. Bull., 1926, 2, 165-180.
- 337. Bridge, E. M., and Iob, L. V. The Mechanism of the Ketogenic Diet in Epilepsy. Amer. J. Psychiat., 1931, 10, 667-671.
- 338. Bridge, E. M., and Iob, L. V. The Mechanism of the Ketogenic Diet in
- Epilepsy. Bull. Johns Hopkins Hosp., 1931, 48, 373-389.
 339. Brock, S. The Problem of the Epilepsies. Internat. Clin., 1928, 4, 38th series, 178-192.
- 340. CAMERON, D. E. The Dehydration Method in Epilepsy. Amer. J. Psychiat., 1931, 11, 123-130.
- 341. CLARK, L. P. The Curability of Idiopathic Epilepsy. Arch. Int. Med., 1912, 9, 1–21.
- 342. CLARK, L. P. Osler's Modern Medicine. 2nd Edition, Vol. 5. New York: Lea and Febiger, 1915. Pp. 592.
- 343. CLARK, L. P. The Nature and Pathogenesis of Epilepsy. New York Med. J., 1915, 101, 385-392, 442-448, 515-522, 567-573, 623-628.
- 344. CLARK, L. P. Is Essential Epilepsy a Life Reaction Disorder? Amer. J. Med. Sci., 1919, 158, 703-711.
- 345. CLARK, L. P. A Psychological Interpretation of Essential Epilepsy. Brain, 1920, 43, 38-49.
- 346. CLARK, L. P. Remarks Upon Consciousness in the Epileptic Fit. Boston Med. and Surg. J., 1921, 185, 494-496.
- 347. CLARK, L. P. A Further Contribution to the Psychology of the Essential Epileptic. J. Nerv. and Ment. Dis., 1926, 63, 575-585.
- 348. CLARK, L. P. Dietetic Treatment of Epileptics. Boston Med. and Surg. J., 1926, **195**, 311–314.
- 349. CLARK, L. P., and CUSHING, K. A Study in Epilepsy. Med. J. and Rec., 1931, 133, 27-31.
- 350. Cohen, M. B., and Lichtig, H. A. Protein Sensitization and Epilepsy. Ohio State Med. J., 1924, 20, 571.
- 351. Collier, J. Epilepsy. Lancet, 1928, 1, 587-591.

- 352. Craig, M. The Early Treatment of Mental Disorder. Chapter XLI in Early Mental Diseases. Lancet, extra numbers, No. 2, 1926(?), 191-
- 353. DAVENPORT, C. B. The Ecology of Epilepsy. Arch. Neurol. and Psychiat., 1923, 9, 554–566.
- 354. Felsen, J. Laboratory Studies in Epilepsy. Arch. Int. Med., 1930, 46, 180-217.
- 355. Fetterman, J. Experiences in the Treatment of Epilepsy. Ohio State Med. J., 1928, 24, 287-290.
- 356. Fox, E. L. Discussion on the Epilepsies. Lancet, 1929, 2, 555.
- 357. GEYELIN, H. R. The Relation of Chemical Influences, Including Diet and Endocrine Disturbances, to Epilepsy. Ann. Int. Med., 1929, 2, 678-681.
- 358. HARPER, W. W. Ketone Diet in Epilepsy. Southern Med. J., 1928, 21, 903-904.
- 359. HELMHOLZ, H. F. The Treatment of Epilepsy in Childhood. J. Amer. Med. Assoc., 1927, 88, 2028-2032.
- 360. HELMHOLZ, H. F., and KEITH, H. M. Eight Years Experience with the Ketogenic Diet in the Treatment of Epilepsy. J. Amer. Med. Assoc., 1930, 95, 707–709.
- 361. HENDERSON, R. C. The Ketogenic Diet Treatment of Epilepsy. U. S. Vet. Bur. Med. Bull., 1930, 6, 122-129.
- 362. Higgins, H. L. Some Physiological and Clinical Effects of High Fat Feeding. New England J. Med., 1930, 203, 145-150.
- 363. Howell, L. P. Epilepsy and Protein Sensitization. Ohio State Med. J., 1923, 19, 660-662.
- 364. Lennox, W. G., and Cobb, S. Epilepsy. Medicine, 1928, 7, 105-290.
- 365. LEVIN, S. J. Allergic Epilepsy: Report of a Case in a Three Year Old Child. J. Amer. Med. Assoc., 1931, 97, 1624-1625.
- 366. McCready, E. B., and Ray, H. M. Allergy as a Factor in the Etiology of Idiopathic Epilepsy. Med. J. and Rec., Supplement, 1924, 120, pp. cxviii-cxix.
- 367. McMurray, T. E. Epilepsy. New York Med. J., 1916, 104, 934.
- 368. McQuarrie, I., and Keith, H. M. Epilepsy in Children. Amer. J. Dis. Child., 1927, 34, 1013-1029.
- 369. Marsh, C. A. A Psychological Theory of the Cause of Epilepsy, with Special Reference to an Abnormal Muscular Expression of a Strong Emotional Drive. Amer. J. Med. Sci., 1920, 159, 450-458.
- 370. Martin, J. P. The Central Nervous Disturbance Manifested by an Epileptic Fit. Lancet, 1926, 1, 760-761.
- 371. MILLER, J. L. Evidence That Idiopathic Epilepsy is a Sensitization
- Disease. Amer. J. Med. Sci., 1924, 168, 635-641.

 372. Orbison, T. J. Psychasthenic Attacks Resembling Epilepsy. Amer. J. Med. Sci., 1910, 140, 392-399.
- 373. Peterman, M. G. The Ketogenic Diet in the Treatment of Epilepsy. Minnesota Med., 1924, 7, 708-711.
- 374. Peterman, M. G. The Ketogenic Diet in Epilepsy. J. Amer. Med. Assoc., 1925, 84, 1979–1983.
- 375. Peterman, M. G. The Ketogenic Diet in the Treatment of Epilepsy. Dietary Admin. and Therapy, 1925, 3, 93-96.
- 376. PETERMAN, M. G. Ketogenic Diet in Epilepsy. Wisconsin Med. J., 1926, **25**, 427–430.
- 377. Peterman, M. G. Epilepsy in Childhood. J. Amer. Med. Assoc., 1927, **88**, 1868–1870.

- 378. Peterman, M. G. The Ketogenic Diet. J. Amer. Med. Assoc., 1928, 90, 1427-1428.
- 379. Pulford, D. S. The Present Status of the Ketogenic Diet. Ann. Int. Med., 1932, 6, 795-801.
- 380. RICHMOND, W. Psychometric Tests in Essential Epilepsy. J. Abnorm. and Soc. Psychol., 1922, 16, 384-391.
- 381. Robertson, A. W. Chronic Intestinal Stasis and Epilepsy. Brit. Med. J., 1924, 2, 1191-1193.
- 382. ROBERTSON, R. C. Epilepsy. U. S. Vet. Bur. Med. Bull., 1926, 2, 848-853.
- 383. Roddis, L. H. Epilepsy as a Protein Sensitization Disease. U. S. Naval Med. Bull., 1925, 23, 15-18.
- 384. Roddis, L. H. The Food History and Protein Sensitization Tests in Epilepsy. U. S. Naval Med. Bull., 1926, 24, 553-556.
- 385. Shanahan, W. T. Some Phases of Epilepsy. Med. J. and Rec., 1931, 134, 166-170.
- 386. SMITH, W. A. The Ketogenic Diet in the Treatment of Epilepsy. Ann. Int. Med., 1929, 2, 1300-1308.
- 387. Spangler, R. H. Allergy and Epilepsy. Analysis of One Hundred Cases. J. Lab. and Clin. Med., 1927-1928, 13, 41-58.
- 388. Spangler, R. H. Some Allergic Factors in Essential Epilepsy. J. Allergy, 1931-1932, 3, 39-50.
- 389. Talbot, F. B. Treatment of Epilepsy. New York: Macmillan Co., 1930. Pp. 308.
- 390. TALBOT, F. B., METCALF, K., and MORIARTY, M. The Ketogenic Diet in the Treatment of Idiopathic Epilepsy. Amer. J. Dis. Child., 1926, 32, 316-320.
- 391. Talbot, F. B., Metcalf, K., and Moriarty, M. E. Epilepsy: Chemical Investigation of Rational Treatment by Production of Ketosis. *Amer. J. Dis. Child.*, 1927, 33, 218-225.
- 392. Talbot, F. B., Metcalf, K. M., and Moriarty, M. E. A Clinical Study of Epileptic Children Treated by Ketogenic Diet. Boston Med. and Sura 1, 1927, 196, 89-96.
- Surg. J., 1927, 196, 89-96.
 393. Tracy, E. A. Diet in the Causation of Epilepsy. Med. J. and Rec., Supplement, 1924, 120, pp. cxv-cxvi.
- 394. Turner, A., Read, S., et al. Discussion on the Nature and Treatment of Epilepsy. Brit. Med. J., 1924, 2, 1045-1054.
- 395. Waldbott, G. L. Allergy as Cause of Epileptiform Convulsions. Arch. Neurol. and Psychiat., 1930, 23, 361-364.
- 396. WALKER, N. P., and WHEELER, G. A. Influence on Epilepsy of a Diet Low in the Pellagra-Preventive Factor. U. S. Pub. Health Rep., 1931, 46, 851-860.
- 397. Wallis, R. L. M., Nicol, W. D., and Craig, M. The Importance of Protein Hypersensitivity in the Diagnosis and Treatment of a Special Group of Epileptics. *Lancet*, 1923, 1, 741-743.
- 398. WARD, J. F. Protein Sensitization as a Possible Cause of Epilepsy and Cancer. New York Med. J. and Med. Rec., 1922, 115, 592-595.
- 399. WARD, J. F., and PATTERSON, H. A. Protein Sensitization in Epilepsy.

 Arch. Neurol. and Psychiat., 1927, 17, 427-443.
- Arch. Neurol. and Psychiat., 1927, 17, 427-443.

 400. WEEKS, D. F., RENNER, D. S., ALLEN, F. M., and WISHART, M. B. Observations on Fasting and Diets in the Treatment of Epilepsy. J. Metab. Res., 1923, 3, 317-364.
- 401. Wiersma, E. T. The Psychology of Epilepsy. Lancet, 1923, 1, 1122-1123.
- 402. WILDER, R. M. The Effect of Ketonemia on the Course of Epilepsy. Mayo Clin. Bull., 1921, 2, 307.

403. WILSON, S. A. K. Discussion on the Epilepsies. Lancet, 1929, 2, 553-554. Also, see Nos. 269, 290, 314 and 319.

Appetite

- 404. Allison, R. S., and Davies, R. P. The Treatment of Functional Anorexia. Lancet, 1931, 1, 902-907.
- 405. BARTLETT, W. M. An Analysis of Anorexia. Amer. J. Dis. Child., 1928, 35, 26-35.
- 406. Benedict, F. G. The Nutritive Requirements of the Body. Amer. J. Physiol., 1906, 16, 409-437.
- 407. Berkman, J. M. Anorexia Nervosa, Anorexia, Inanition, and Low Basal Metabolic Rate. Amer. J. Med. Sci., 1930, 180, 411-424.
- 408. Berman, L. Food and Character. New York: Houghton Mifflin & Co., 1932. Pp. 368.
- 409. Burack, E., and Cowgill, G. R. Anorexia Characteristic of Lack of the Vitamin B Complex: The Rôles of the Individual Components. Proc. Soc. Exper. Biol. and Med., 1931, 28, 750-752.
- 410. BUTLER, T. J. The Psychologic Treatment of Loss of Appetite in Children. Pennsylvania Med. J., 1929, 32, 608-609.
- 411. CARLSON, A. J. The Control of Hunger in Health and Disease. Chicago: Univ. of Chicago Press, 1916. Pp. 319.
- 412. CARPENTER, W. B. Mental Dietetics: The Effects of Stimulants, Solid and Fluid, on the Mind. J. Psychol. Med. and Ment. Path., 1851, 4, 89-111.
- 413. CHITTENDEN, R. H. The Nutrition of Man. New York: Frederick A. Stokes Co., 1907. Pp. 321.
- 414. Cowgill, G. R. A Contribution to the Study of the Relationship Between Vitamin-B and the Food Intake in the Dog. *Proc. Soc. Exper. Biol. and Med.*, 1920-1921, 18, 290-291.
- 415. Cowgill, G. R. A Contribution to the Study of the Relation Between Vitamin-B and the Nutrition of the Dog. Amer. J. Physiol., 1921, 57, 420-436.
- 416. Cowgill, G. R. An Improved Procedure for Metabolism Experiments. J. Biol. Chem., 1923, 56, 725-737.
- 417. Cowgill, G. R. Recent Studies in the Physiology of Vitamin B. Yale J. Biol. and Med., 1928–1929, 1, 353–361.
- 418. Cowgill, G. R. The Energy Factor in Relation to Food Intake: Experiments on the Dog. Amer. J. Physiol., 1928, 85, 45-64.
- 419. Cowgill, G. R., Deuel, H. J., and Smith, A. H. Studies in the Physiology of Vitamins. III. Quantitative Aspects of the Relation Between Vitamin B and Appetite in the Dog. Amer. J. Physiol., 1925, 73, 106-126
- 420. Cowgill, G. R., Rosenberg, H. A., and Rogoff, J. Studies in the Physiology of Vitamins. *Amer. J. Physiol.*, 1930, 95, 537-541.
- 421. CRICHTON-BROWN, J. Parcimony in Nutrition. New York: Funk and Wagnalls Co., 1909.
- 422. Davis, C. M. Self Selection of Diet by Newly Weaned Infants. Amer. J. Dis. Child., 1928, 36, 651-679.
- 423. Dennett, R. H. Routine Use of the Vitamin B Factor in Infant Feeding. J. Amer. Med. Assn., 1929, 92, 769-772.
- 424. Drummond, J. C. A Study of the Water-Soluble Accessory Growth Promoting Substance. II. Its Influence Upon the Nutrition, and Nitrogen Metabolism of the Rat. Biochem. J., 1918, 12, 25-41.

- 425. DRUMMOND, J. C., and MARRIAN, G. F. The Relation of Vitamin B to Tissue Oxidations. *Biochem. J.*, 1926, 20, 1229-1255.
- 426. DUNLAP, K. Instinct and Desire. J. Abnorm. and Soc. Psychol., 1925, 20, 170-173.
- 427. Evvard, J. M. Is the Appetite of Swine a Reliable Indication of Physiological Needs? Proc. Iowa Acad. Sci., 1915, 22, 375-403.
- 428. Fisher, I. The Effect of Diet on Endurance. New Haven, Connecticut: Yale Univ. Press, 1918.
- 429. GAUGER, M. E. The Modifiability of Response to Taste-Stimuli in the Preschool Child. Teachers College, Columbia Univ., Contrib. to Educ., No. 348, 1929. Pp. 53.
- 430. GIRDEN, E. S. Cannibalism in Dogs. J. Comp. Psychol., 1932, 14, 409-413.
- 431. Graham, C. E., and Griffith, W. H. Some Effects of the Vitamin B Complex on Appetite and on Utilization of Food. *Proc. Soc. Exper. Biol. and Med.*, 1931, 28, 1086-1088.
- 432. GREEN, H. H. Perverted Appetites. Physiol. Rev., 1925, 5, 336-348.
- 433. Gross, L. The Effects of Vitamin Deficient Diets on Rats, with Special Reference to the Motor Functions of the Intestinal Tract in Vivo and Vitro. J. Path. and Bact., 1924, 27, 27-50.
- 434. HAUSMANN, M. F. The Behavior of Albino Rats in Choosing Food and Stimulants. J. Comp. Psychol., 1932, 13, 279-309.
- 435. Holder, R. C., Smith, C. A., and Hawk, P. B. Is Unpalatable Food Properly Digested? Science, 1920, 51, 299.
- 436. Hoobler, B. R. Symptomatology of Vitamin B Deficiency in Infants. J. Amer. Med. Assoc., 1928, 91, 307-310.
- 437. Howell, W. H. Text-Book of Physiology. 10th Edition. Philadelphia: W. B. Saunders Co., 1927.
- 438. KARR, W. G. Some Effects of Water-Soluble Vitamine upon Nutrition. J. Biol. Chem., 1920, 44, 255-276.
- 439. Kemp, M. Unbalanced Diet as a Cause of Poor Appetite in Children. Pennsylvania Med. J., 1929, 32, 607-608.
- 440. Kerley, C. G. Apparent and Real Appetite Defects in the Young. Med. Clin. North Amer., 1917, 1, 505-512.
- 441. Kerley, J. H. Hyperchlorhydria—A Frequent Cause of Defective Appetite in Children. Med. Rec., 1920, 97, 786-787.
- 442. Kon, S. K. The Self-Selection of Food Constituents by the Rat. Biochem. J., 1931, 25, 473-481.
- 443. Kon, S. K., and Drummond, J. C. Study of Vitamin B Deficiency in Pigeons. *Biochem. J.*, 1927, 21, 632-652.
- 444. Kugelmass, I. N., and Samuel, E. L. Raw Basic Feeding in Anorexia of Childhood. Arch. Ped., 1931, 48, 457-462.
- 445. Levine, V. E. Why We Should Be More Interested in Nutrition. Dietary Admin. and Therapy, 1925, 3, 465-479.
- 446. Light, R. F., Miller, G., and Frey, C. N. Studies on the Effects of Overdosage of Vitamin D. J. Biol. Chem., 1929, 84, 487-494.
- 447. Loeb, J. Forced Movements, Tropisms and Animal Conduct. Philadelphia: J. B. Lippincott Co., 1918.
- 448. Lucas, W. P., and Pryor, H. B. Factors Involved in Combating the "Hunger Strike" in Children. Amer. J. Dis. Child., 1931, 41, 249-261.
- 449. McCandlish, A. C. Appetite as a Guide in Feeding Dairy Calves. Iowa Agri. Exper. Sta. Res. Bull., No. 51, 1919. Pp. 177-184.
- 450. McCarrison, R. Studies in Deficiency Disease. London: Frowde, Hodder and Stoughton, 1921. Pp. 270.

- 451. McClendon, J. F. Nutrition and Public Health with Special Reference to Vitamines. Amer. J. Med. Sci., 1920, 159, 477-497.
- 452. McCollum, E. V. Nuclein Synthesis in the Animal Body. Amer. J. Physiol., 1909, 25, 120-141.
- 453. McCollum, E. V., Simmonds, N., and Pitz, W. The Vegetarian Diet in the Light of Our Present Knowledge of Nutrition. Amer. J. Physiol., 1916, 41, 333-360.
- 454. McLaughlin, L., Tarwater, M., Lowenberg, M., and Koch, G. Vegetables in the Diets of Preschool Children. J. Nutrition, 1931, 4, 115-125.
- 455. Maslow, A. H. The "Emotion" of Disgust in Dogs. J. Comp. Psychol., 1932, 14, 401-407.
- 456. MITCHELL, H. S., and MENDEL, L. B. Studies in Nutrition: The Choice Between Adequate and Inadequate Diet, as Made by Rats and Mice. Amer. J. Physiol., 1921, 58, 211-225.
- 457. Mursell, J. L. Contributions to the Psychology of Nutrition. I. Hunger and Appetite. *Psychol. Rev.*, 1925, 32, 317-333.
- 458. NEVENS, W. B. Experiments in the Self-Feeding of Dairy Cows. Illinois Agri. Exper. Sta. Bull., No. 289, 1927. Pp. 425-452.
- 459. OSBORNE, T. B. What and How Much Should We Eat. Atlantic Monthly, 1918, 122, 332-341.
- 460. OSBORNE, T. B. The Chemistry of Nutrition. Boston Med. and Surg. J., 1919, 181, 77-78. (Letter to the Editor.)
- 461. OSBORNE, T. B., and MENDEL, L. B. Feeding Experiments with Isolated Food-Substances. Bull. No. 156, Parts I and II, Carnegie Institution of Washington, Washington, D. C., 1911.
- of Washington, Washington, D. C., 1911.

 462. Osborne, T. B., and Mendel, L. B. The Choice Between Adequate and Inadequate Diets, as Made by Rats. J. Biol. Chem., 1918, 35, 19-27.
- 463. OSBORNE, T. B., and MENDEL, L. B. The Rôle of Vitamines in the Diet. J. Biol. Chem., 1917, 31, 149-159.
- 464. RICE, C. H. Relation of Acquired Food Dislikes of Childhood to Diseases of Middle Life. J. Amer. Med. Assoc., 1920, 75, 100-102.
- 465. Rudolfs, W., and Lackey, J. B. Effect of Food Upon Phototropism of Mosquito Larvae. Amer. J. Hygiene, 1929, 10, 245-252.
- 466. SCHULTZ, F. W. The Problem of Chronic Anorexia in Childhood. J. Amer. Med. Assoc., 1930, 94, 73-77.
- 467. SHERMAN, H. C. Chemistry of Food and Nutrition. New York: The Macmillan Co., 1923. Pp. 454.
- 468. SHERMAN, H. C., and SANDELS, M. R. Further Experimental Differentiation of Vitamins B and G. J. Nutrition, 1931, 3, 395-409.
- 469. SLONAKER, J. R., and CARD, T. A. The Effect of a Restricted Diet. V. On Mortality, Cannibalism and the Sex Ratio. Amer. J. Physiol., 1923, 64, 297-310.
- 470. STUCKY, C. J., and Rose, W. B. Studies in the Physiology of Vitamins. Amer. J. Physiol., 1929, 89, 1-17.
- 471. Sure, B. Vitamin Requirements of Nursing Young. J. Nutrition, 1928, 1, 155-164.
- 472. Sure, B. A Detailed Study of the Rôle of Vitamin B in Anorexia in the Albino Rat. J. Nutrition, 1928, 1, 49-56.
- 473. West, J. H. Alterations in the Mechanics of the Gastro-Intestinal Tract as a Cause of Poor Appetite in Children. *Pennsylvania Med. J.*, 1929, 32, 609-611.
- 474. WILEY, H. W. The Relation of Food to Health and Vitality. Internat. Clin., 1925, 2, 35th series, 209-211.

- 475. Wright, S. The Effect of B-Vitamin on the Appetite. Lancet, 1921, 2, 1208-1209.
- 476. Young, P. T. Preferential Discrimination of the White Rat for Different Kinds of Grain. Amer. J. Psychol., 1928, 40, 372-400.
- 477. Young, P. T. Relative Food Preferences of the White Rat. J. Comp. Psychol., 1932, 14, 297-319.

Racial Vigor and Temperament

- 478. Adolph, W. H. Aspects of Nutrition and Metabolism in China. Sci-Monthly, 1929, 29, 39-43.
- 479. Armitage, F. P. Diet and Race. New York: Longmans, Green & Co., 1922.
- 480. BENEDICT, F. G., and CARPENTER, T. M. Food Ingestion and Energy Transformations. Carnegie Institution of Washington, Washington, D. C., 1918. Pub. No. 261.
- 481. Benedict, F. G., Miles, W. R., Roth, P., and Smith, H. M. Human Vitality and Efficiency Under Prolonged Restricted Diet. Carnegie Institution of Washington, Washington, D. C., 1919. No. 280.
- 482. Benedict, F. G., and Roth, P. The Metabolism of Vegetarians as Compared with the Metabolism of Non-Vegetarians of Like Weight and Height. J. Biol. Chem., 1915, 20, 231-241.
- Height. J. Biol. Chem., 1915, 20, 231-241.

 483. Chun, J. W. H. The Influence of the Chinese Diet on Disease. China Med. J., 1925, 39, 1056-1049.
- Med. J., 1925, 39, 1056-1049.

 484. Conger, R. M. The Effect of Diet on Activity. Master's Thesis, Iowa State College, Ames, Iowa, 1929.
- 485. CRILE, G. W. The Kinetic Drive. J. Amer. Med. Assoc., 1915, 65, 2129-2138.
- 486. Du Bois, E. F. The Control of Protein in the Diet. J. Amer. Diet. Assoc., 1928, 4, 53-76.
- 487. Grayson, T. W. The Best Diet. Internat. Clin., 1927, 1, 37th series, 114-119.
- 488. Heinbecker, P. Studies on the Metabolism of Eskimos. J. Biol. Chem., 1928, 80, 461-475.
- 489. HITCHCOCK, F. A. The Effect of the Luxus Consumption of Meat upon the Voluntary Activity and Growth of the Albino Rat. Amer. J. Physiol., 1926, 79, 206-217.
- 490. HITCHCOCK, F. A. The Effect of Low Protein and Protein-Free Diets and Starvation on the Voluntary Activity of the Albino Rat. Amer. J. Physiol., 1928, 84, 410-416.
- 491. Holck, H. H. G. Diet and Efficiency. Chicago: University of Chicago Press, 1929. Pp. 72.
- 492. Holt, L. E. Food, Health and Growth. New York: Macmillan Co., 1925. Pp. 273.
- 493. Lieb, C. W. The Effects of an Exclusive, Long-Continued Meat Diet. J. Amer. Med. Assoc., 1926, 87, 25-26.
- 494. Lieb, C. W. The Effects on Human Beings of a Twelve Months' Exclusive Meat Diet. J. Amer. Med. Assoc., 1929, 93, 20-22.
- 495. Liebig, Baron. The Nutritive Value of Different Sorts of Food. Lancet, 1869, 1, 186-187.
- 496. LORAND, A. Health and Longevity Through Rational Diet. Philadelphia: F. A. Davis Co., 1928. Pp. 432.
- 497. Lusk, G. The Specific Dynamic Action of Various Food Factors. *Medicine*, 1922, 1, 311-354.

- 498. McClellan, W. S. The Effect of the Prolonged Use of Exclusive Meat Diets on Two Men. J. Amer. Diet. Assoc., 1930, 6, 216-228.
- 499. McCollum, E. V. The Newer Knowledge of Nutrition. 2nd Edition. New York: Macmillan Co., 1923.
- 500. McCollum, E. V., Simmonds, N., and Parsons, H. T. A Biological Analysis of Pellagra-Producing Diets. J. Biol. Chem., 1919, 38, 113-146.
- 501. MACLEOD, G., CROFTS, E. E., and BENEDICT, F. G. The Basal Metabolism of Some Orientals. Amer. J. Physiol., 1925, 73, 449-462.
- 502. Mason, C. C. German Nutrition, 1914-1919. Bull. Johns Hopkins Hosp., 1920, 31, 66-79.
- 503. Mason, E. D., and Benedict, F. G. The Basal Metabolism of South Indian Women. Indian J. Med. Res., 1931, 19, 75-98.
- 504. Mendel, L. B. Some Historical Aspects of Vegetarianism. Pop. Sci. Monthly, 1904, March, 457.
- 505. MINOT, G. R. Some Fundamental Clinical Aspects of Deficiencies. Ann. Int. Med., 1929, 3, 216-229.
- 506. MUKHERJEE, H. N., and GUPTA, P. C. The Basal Metabolism of Indians (Bengalis). Indian J. Med. Res., 1931, 18, 807-812.
- 507. von MÜLLER, F. Observations During the Period of Under-Nutrition in Germany. Bull. New York Acad. Med., 1926, 2, 502-516. (Abstract in Bull. Hygiene, 1927, 2, 796-797.)
- 508. MURLIN, J. R. Some Unsolved Problems in the Nutrition of Athletes. Amer. Physical Educ. Rev., 1926, 31, 814-821.
- 509. NECHELES, H. Basal Metabolism in Orientals. Amer. J. Physiol., 1930, 91, 661-663.
- 510. OSHIMA, K. A Digest of Japanese Investigations on the Nutrition of Man. U. S. Dept. Agri., Office Exper. Sta., Bull. No. 159, 1905. Pp. 224.
- 511. SHERMAN, H. C. Nutrition and Vigor. Amer. Physical Educ. Rev., 1923, 28, 462-469.
- 512. SLONAKER, J. R. The Effect of a Strictly Vegetable Diet on the Spontaneous Activity, the Rate of Growth, and the Longevity of the Albino Rat. Leland Stanford Junior University Pub., 1912.
- 513. SLONAKER, J. R. The Effect of Pubescence, Oestruation and Menopause on the Voluntary Activity in the Albino Rat. Amer. J. Physiol., 1924, 68, 294-315.
- 514. SLONAKER, J. R. Effect of Different Per Cents of Protein in the Diet. II. Spontaneous Activity. Amer. J. Physiol., 1931, 96, 557-561.
- 515. STERN, F. The Nutritionist Looks at Mental Hygiene. Ment. Hygiene, 1930, 14, 54-66.
- 516. TANG, Y., CHIN, K., and TSANG, Y. H. The Effect of a Vegetarian Diet on the Learning Ability of Albino Rats. Contrib. Nat. Res. Instit. Psychol., Acad. Clinica, 1932, 1, 1-27.
- 517. Tilt, J. The Basal Metabolism of Young College Women in Florida. J. Biol. Chem., 1930, 86, 635-641.
- 518. TREMBLY, E. R. Influence of a Ketogenic Diet on Growth, Activity and Blood Picture of the Albino Rat. Master's thesis, Iowa State College, Ames, Iowa, 1929.
- 519. WAKEHAM, G., and HANSEN, L. A. The Basal-Metabolic Rates of Vegetarians. Science, 1931, 74, 70-71.
- 520. WAKEHAM, G., and HANSEN, L. O. The Basal Metabolic Rates of Vegetarians. J. Biol. Chem., 1932, 97, 155-162.

- 521. Weston, W. The Food Elements as Factors in Preventive Medicine. J. South Carolina Med. Assoc., 1927, 23, 489-495.
- 522. WHERRY, E. T. Does a Low-Protein Diet Produce Racial Inferiority? Science, 1913, 37, 908-909.
- 523. WHITACRE, J., and BLUNT, K. Coefficient of Digestibility and Dynamic Action of a Simple Diet in Contrasting Types of Individuals. J. Home Ec., 1927, 19, 20-27.
- 524. WISHART, G. M. The Influence of the Protein Intake on the Basal Metabolism. J. Physiol., 1928, 65, 243-254.
- 525. Wu, H., and Chen, T. T. Basal Metabolism of Omnivorous and Vegetarian Rats. Chinese J. Physiol., 1929, 3, 315-317.
- 526. Wu, H., and Wu, D. Y. Growth of Rats on Vegetarian Diets. *Chinese J. Physiol.*, 1928, 2, 173-194.

 Also, see Nos. 160, 406, 408, 412, and 413.

Endurance

- 527. Bassett, S. H., Holt, E., and Santos, F. O. The Influence of Meat upon Physical Efficiency. Amer. J. Physiol., 1922, 60, 574-577.
- 528. BERRY, E. The Effects of a High and Low Proteid Diet on Physical Efficiency. Amer. Physical Educ. Rev., 1909, 14, 288-297.
- 529. Chapin, W. H. Diet—the Proteids. Amer. Physical Educ. Rev., 1907, 12, 121-125.
- 530. FISHER, I. The Influence of Flesh Eating on Endurance. Yale Med. J., 1907, 13, 205-221.
- 531. FISHER, I. Diet and Endurance at Brussels. Science, 1907, 26, 561-563.
- 532. FISHER, I. Influence of Flesh Eating on Endurance. J. Sci. Phys. Trg., 1914, 6, 98-106.
- 533. HINDHEDE, M. Diet and Health. Practitioner, 1926, 116, 249-261.
- 534. HINDHEDE, M. The Biological Value of Bread-Protein. Biochem. J., 1926, 20, 330-334.
- 535. Howe, E. C. Diet and Endurance. Amer. Physical Educ. Rev., 1916, 21, 490-502.
- 536. Krogh, A., and Lindhard, J. The Relative Value of Fat and Carbohydrate as Sources of Muscular Energy. *Biochem. J.*, 1920, 14, 290-363.
- 537. LAIRD, D. A. The Effects of Sugar in Recovering Mental and Motor Control After Brief Periods of Exercise. Med. Rev. of Reviews, 1930, 36, 383-386.
- 538. Langworthy, C. F. A Strength and Endurance Test. Science, 1911, 33, 708-711.
- 539. Nelson, V. E., Baldwin, F. M., Riggs, A. G., and Cunningham, M. The Relation of Vitamin Deficiency to Muscle Fatigue. Amer. J. Physiol., 1925, 72, 69-75.
- 540. Peters, R. A. Co-Ordinative Bio-Chemistry of the Cell and Tissues. J. State Med., 1930, 38, 63-87.
- 541. Talbot, F. B. Fatigue—The American Disease. Proc. Inter-state Post Grad. Med. Assemb. North Amer., 1927, pp. 114-118.
- 542. Thompson, W. G. Practical Dietetics. New York: D. Appleton & Co., 1905. Pp. 846.
 - Also, see Nos. 408, 428, 481, 487, 491, 496, and 502.

Intelligence and Learning

543. Anderson, J. E., and Smith, A. H. Effect of Quantitative and Qualitative Stunting upon Maze Learning in the White Rat. J. Comp. Psychol., 1926, 6, 337-359.

544. Anderson, J. E., and Smith, A. H. Relation of Performance to Age and Nutritive Condition in the White Rat. J. Comp. Psychol., 1932, 13, 409-446.

545. BALYEAT, R. M. The Hereditary Factor in Allergic Disease. With Special Reference to the General Health and Mental Activity of Allergic Patients. *Amer. J. Med. Sci.*, 1928, 176, 332-345.

546. BALYEAT, R. M. The General Health and Mental Activity of Allergic Children. Amer. J. Dis. Child., 1929, 37, 1193-1197.

547. Blanton, S. Mental and Nervous Changes in the Children of the Volksschulen of Trier, Germany, Caused by Malnutrition. *Ment. Hygiene*, 1919, 3, 343-386.

548. BLISS, D. C. Malnutrition, a School Problem. Elementary School J., 1921, 21, 515-521.

549. CRAMER, W. On Vitamin Underfeeding. Brit. J. Exper. Path., 1922, 3, 298-306.

550. Cramer, W. Vitamins and the Borderland Between Health and Disease. Lancet, 1924, 1, 633-640.

551. Down, H. L. Relations of Mental Retardation to Nutrition. Hosp. Soc. Service, 1922, 6, 92-95.

552. Editorial, Diet and Intelligence. Amer. Med., 1931, 37, 109-110.

553. Editorial, The Health and the Intelligence and Physique of School Children. Brit. Med. J., 1923, 2, 334.

dren. Brit. Med. J., 1923, 2, 334.

554. Frank, M. The Effect of a Rickets-Producing Diet on the Learning Ability of White Rats. J. Comp. Psychol., 1932, 13, 87-105.

555. Fritz, M. F. Maze Performance of the White Rat in Relation to Unfavorable Salt Mixture and Vitamin B Deficiency. J. Comp. Psychol., 1932, 13, 365-390.

556. GESELL, A. Infancy and Human Growth. New York: Macmillan Co., 1928. Pp. 418.

557. Graper, F. M., and Park, E. W. The Effect of Improved Feeding on the Physical and Mental Development of Under-Nourished and Backward Children. J. Home Ec., 1923, 15, 627-632.

558. Hoefer, C., and Hardy, M. C. Later Development of Breast Fed and Artificially Fed Infants. J. Amer. Med. Assoc., 1929, 92, 615-619.

559. Hoobler, B. R., Outhouse, J., and Macy, I. G. Certain Biological Properties of Human Milk. Trans. Amer. Pediat. Soc., 1926, 38, 38-40.

560. Hunt, J. L., Johnson, B. J., and Lincoln, E. M. Health Education and the Nutrition Class. New York: E. P. Dutton and Co., 1921. Pp. 281.
 561. Levine, V. E. Some Fallacies with Reference to Nutrition. Dietary

561. Levine, V. E. Some Fallacies with Reference to Nutrition. Dietary Admin. and Therapy, 1925, 3, 547-556.

562. LEVINE, V. E. The Importance of Nutrition in Child Hygiene. Sci. Monthly, 1929, 28, 554-559.

563. McCollum, E. V. Fundamentals of Nutrition. Internat. Clin., 1932, 2, 42nd series, 1-17.

564. Macy, I. G., Outhouse, J., Graham, A., and Long, M. L. Human Milk Studies. III. The Quantitative Estimation of Vitamin B. J. Biol. Chem., 1927, 73, 189-201.

565. MAURER, S., and HANKE, M. T. Diet, Health and Intelligence. Illinois Health Quart., 1931, 3, 214-223.

- 566. MAURER, S., and TSAI, L. S. Vitamin B Deficiency in Nursing Young Rats and Learning Ability. Science, 1929, 70, 456-458.
- Rats and Learning Ability. Science, 1929, 70, 456-458.

 567. MAURER, S., and TSAI, L. S. Vitamin B Deficiency and Learning Ability.

 J. Comp. Psychol., 1930, 11, 51-62.
- 568. MAURER, S., and TSAI, L. S. Diet and Intelligence. Illinois Health Quart., 1930, 2, 131-135.
- 569. MAURER, S., and TSAI, L. S. The Effect of Partial Depletion of Vitamin B Complex upon Learning Ability in Rats. J. Nutrition, 1931, 4, 507-516.
- 570. Moore, C. U., Brodie, J. L., and Hope, R. B. Some Effects upon the Young of Inadequate Maternal Diets. Amer. J. Physiol., 1927, 82, 350-357.
- 571. NICHOLLS, E. E. Performances in Certain Mental Tests of Children Classified as Underweight and Normal. J. Comp. Psychol., 1923, 3, 147-179.
- 572. Peters, A. Relation of Biochemistry to the Problems of Psychopathology. J. Psycho-Asthenics, 1912, 17, 56-61.
- 573. Rosenberg, L. C. Malnutrition in Children. Amer. J. Dis. Child., 1931, 41, 303-336.
- 574. Ruch, F. L. The Effect of Inanition Upon Maze Learning in the White Rat. J. Comp. Psychol., 1932, 14, 321-329.
- 575. SMITH, A. J., and FIELD, A. M. A Study of the Effect of Nutrition on Mental Growth. J. Home Ec., 1926, 18, 686-690.
- 576. STALNAKER, E. M. A Comparison of Certain Mental and Physical Measurements of School Children and College Students. J. Comp. Psychol., 1923, 3, 181-239.
 - Also, see Nos. 423, 481, 491, 492, and 516.

Mental Disorders

- 577. APPEL, K. E., and FARR, C. B. The Specific Dynamic Action of Protein in Relation to Mental Disease. J. Nerv. and Ment. Dis., 1929, 70, 43-50.
- 578. Bostock, J. Digestion and Mental Disease: An Analysis of One Hundred Consecutive Fractional Test Meals with Some Animal Experiments. Med. J. Australia, 1926, 1, 510-517.
- 579. Bronfenbrenner, A. N. Glucose as a Factor in Metabolism. Proc. Soc. Exper. Biol. and Med., 1926-1927, 24, 269-273.
- 580. BRYANT, J. Chronic Fatigue: Diet, Exercise, and Other Factors in Treatment. Boston Med. and Surg. J., 1920, 182, 629-631.
- 581. Buckley, C. W. The Treatment of Neurasthenia by Rest, Diet, and Massage. *Practitioner*, 1911, 86, 140-150.
- 582. CARTER, H. S., Howe, P. E., and Mason, H. H. Nutrition and Clinical Dietetics. 2nd Edition. Philadelphia: Lea and Febiger, 1921. Pp. 703.
- 583. Firch, W. E. Dietotherapy. Vol. III. New York: D. Appleton & Co., 1918. Pp. 929.
- 584. GREGG, D. Avitaminotic Depressions. New England J. Med., 1929, 201, 420-422.
- 585. HARTWELL, G. A., and MOTTRAM, V. H. The Brown v. White Bread
- Controversy. Lancet, 1929, 2, 892-894.

 586. Henry, G. W. Emotions and Digestive Functions. J. Amer. Dietetic Assoc., 1927-1928, 3, 19-23.
- 587. HUGHES, J. S., AUBEL, C. E., and LIENHARDT, H. F. The Importance of Vitamin A and Vitamin C in the Ration of Swine. Agri. Exp. Sta., Kans. State Agr. College Tech. Bull., 23, June, 1928.

- 588. Hughes, J. S., Lienhardt, H. F., and Aubel, C. E. Nerve Degeneration Resulting from Avitaminosis A. J. Nutrition, 1929, 2, 183-186.
- 589. LAIRD, D. A., LEVITAN, M., and WILSON, V. A. Nervousness in School Children as Related to Hunger and Diet. Med. J. and Rec., 1931, 134, 494-499.
- 590. McCarrison, R. The Pathogenesis of Deficiency Disease. VIII. The General Effects of Deficient Dietaries on Monkeys. Indian J. Med. Res., 1919-1920, 7, 308-341.
- 591. MERCIER, C. Diet as a Factor in the Causation of Mental Disease. Lancet, 1916, 1, 510–513, 561–565.
- 592. PATON, D. N. Neurasthenia and Diet. Practitioner, 1911, 86, 122-132.
- 593. RICHARDSON, H. K. The Relationship of Mental Disease to Digestion and Nutrition. J. Amer. Dietetic Assoc., 1928-1929, 4, 228-236.
- 594. SEHAM, M., and SEHAM, G. The Relation Between Malnutrition and Nervousness. Amer. J. Dis. Child., 1929, 37, 1-38.
- 595. SHERRILL, J. W. Metabolic Observations in Psychiatric Cases. J. Metab.
- Res., 1924, 5, 129-143.
 596. Thomas, W. R. Dementia Praecox and Vitamins. J. Ment. Sci., 1928, 74, 460–464.
- 597. TIMME, W. Some Clinical Aspects of a Diminished Calcium Utilization. Internat. Clin., 1931, 4, 41st series, 166-174.
- 598. WALSH, J. J. Neuroses and Psychoneuroses and the Therapeutic Value of Food. Internat. Clin., 1921, 3, 31st series, 156-174.
- 599. WALSH, J. J. Eating as a Therapeutic Measure. Internat. Clin., 1932, 2, 42nd series, 95-117. Also, see Nos. 450, 487, 492, 496, and 562.

Intestinal Toxemia

- 600. ALVAREZ, W. C. Intestinal Autointoxication. Physiol. Rev., 1924, 4, 352-393.
- 601. Andrewes, F. W. The Bacteriology of the Alimentary Canal. Proc. Royal Soc. Med., 1913, 6, 11-20.
- 602. Bartle, H. J. Protein Intoxication. I. Enigmas and Axioms of Protein Digestion, Metabolism, Elimination. II. Clinical Study of Fifty Cases. III. Treatment. Med. J. and Rec., 1928, 128, 28-32, 63-66, 386-389, 446-448.
- 603. Boles, R. S. A Consideration of Intestinal Toxemia with Especial Reference to the Use of Colonic Irrigations. Med. Clin. North Amer., 1924, **8**, 845–863.
- 604. Cotton, H. A. Infection of Gastrointestinal Tract in Relation to Systemic
- Disorders. Amer. J. Med. Sci., 1922, 164, 329-338.
 605. CRILE, G. W. Studies in Exhaustion: VII. Autointoxication. Surg., 1924, 9, 293-308.
- 606. DIAMOND, J. S. Gastrointestinal Neuroses and Their Management. Med. J. and Rec., 1931, 134, 476-481.
- 607. Donaldson, A. N. Relation of Constipation to Intestinal Intoxication. J. Amer. Med. Assoc., 1922, 78, 884–888.
- 608. FITCH, W. E. Dietotherapy. Vol. III. Pp. 113-122. New York: D. Appleton & Co., 1918. Pp. 929.
- 609. FITCH, W. E. Putrefactive Intestinal Toxemia. Med. J. and Rec., 1930, 132, 183-187.
- 610. GANT, S. G. Constipation, Obstipation and Intestinal Stasis. delphia: W. B. Saunders Co., 1916. Pp. 584.

- 611. GRoss, L. The Effects of Vitamin Deficient Diets on Rats, with Special Reference to the Motor Functions of the Intestinal Tract in Vivo and in Vitro. J. Path. and Bact., 1924, 27, 27-50.
- 612. KAUFFMAN, O. J. Gastro-Intestinal Auto-Intoxication as a Factor in Nervous Disorders. Proc. Inter-State Post Grad. Med. Assembly North Amer., 1927, pp. 357-361.
- 613. Kellogg, J. H. Colon Hygiene. Battle Creek, Michigan: Good Health Pub. Co., 1915. Pp. 393.
- 614. Kellogg, J. H. Autointoxication or Intestinal Toxemia. Battle Creek, Michigan: Modern Med. Pub. Co., 1919. Pp. 366.
- 615. Kraetzer, A. F. Neurasthenia. Med. Clin. North Amer., 1927, 10, 1051-1061.
- 616. PAULSEN, A. E. The Influence of Treatment for Intestinal Toxemia on Mental and Motor Efficiency. Arch. Psychol., 1924, No. 69, pp. 45.
- Mental and Motor Efficiency. Arch. Psychol., 1924, No. 69, pp. 45. 617. SATTERLEE, G. R. Infection of the Gastrointestinal Trace in Systemic Disorders. Amer. J. Med. Sci., 1922, 164, 313-322.
- 618. SATTERLEE, G. R., and ELDRIDGE, W. W. Symptomatology of the Nervous System in Chronic Intestinal Toxemia. J. Amer. Med. Assoc., 1917, 69, 1414-1418.
- 619. SAUNDBY, R. The Consequences and Treatment of Alimentary Toxaemia from a Medical Point of View. Proc. Royal Soc. Med., 1913, 6, 37-48.
- 620. STUCKY, J. A. Deficient Vitamin Diets as a Factor in Otolaryngological Conditions. Laryngoscope, 1924, 34, 647-654.
- 621. Synnorr, M. J. Intestinal Toxemia, Its Diagnosis and Treatment. Med.
- J. and Rec., 1932, 136, 441-447.
 622. WALKER, J. Functional Mental Disorder Due to Toxaemia from the Bowel. Lancet, 1924, 2, 1058-1060.
- 623. WILE, I. S. Constipation and Behavior. Amer. J. Dis. Child., 1929, 38, 570-589.
- 624. YEARSLEY, M. Intestinal Toxaemic Deafness in Children. Brit. J. Child. Dis., 1929, 26, 116-120.
 Also, see Nos. 321 and 352.

Sense Organs

- 625. Adler, F. H. Ocular Disorders in Deficiency Diseases. Arch. Ophthal., 1927, 56, 593-611.
- 626. AYKROYD, W. R. Vitamin A Deficiency in Newfoundland. Irish J. Med. Sci., 1928, 28, 161.
- 627. AYKROYD, W. R. Beriberi and Other Food-Deficiency Diseases in Newfoundland and Labrador. J. Hygiene, 1930, 30, 357-386.
- 628. BARLOW, R. A. Does Vitamin-Deficient Diet Cause Deafness? Results of Animal Experimentation. Laryngoscope, 1927, 37, 640-648.
- 629. Bloch, C. E. Blindness and Other Diseases in Children Arising from Deficient Nutrition (Lack of Fat-Soluble A Factor). Amer. J. Dis. Child., 1924, 27, 139-148.
- 630. Bordley, J. A Family of Hemeralopes. Johns Hopkins Hosp. Bull., 1908, 19, 278-281.
- 631. Enright, J. I. War Oedema in Turkish Prisoners of War. Lancet, 1920, 1, 314-316.
- 632. Fredericia, L. S., and Holm, E. Experimental Contribution to the Study of the Relation Between Night Blindness and Malnutrition. Amer. J. Physiol., 1925, 73, 62-78.

- 633. Holm, E. Demonstration of Hemeralopia in Rats Nourished on Food Devoid of Fat-Soluble-A-Vitamin. Amer. J. Physiol., 1925, 73, 79-84.
- 634. LITTLE, J. M. Beriberi Caused by Fine White Flour. J. Amer. Med. Assoc., 1912, 58, 2029-2030.
- 635. Peterson, F. Nutrition and Nerves. Arch. Neurol. and Psychiat., 1925, 14, 435-439.
- 636. PILLAT, A. The Frequency of Deficiency Diseases of the Eye Due to Lack of Vitamin A in a Military Camp North of Peiping. Nat. Med. J. China, 1929, 15, 585-591.
- 637. SMITH, H. Night Blindness and the Malingering of Night Blindness. J. Amer. Med. Assoc., 1921, 77, 1001-1002.
- 638. Spence, J. C. A Clinical Study of Nutritional Xerophthalmia and Night-Blindness. Arch. Dis. in Childhood, 1931, 6, 17-26.
- 639. Spivacke, C. A. Constitutional Allergic Reactions. Med. J. and Rec., 1930, 131, 447-448.
- 640. Tansley, K. The Regeneration of Visual Purple: Its Relation to Dark Adaptation and Night Blindness. J. Physiol., 1931, 71, 442-458.
- 641. Weston, W. The Effect of the Food Elements Upon the Special Senses. Ann. Otol., Rhin. and Laryn., 1928, 37, 618-626.

Allergy

- 642. Andresen, A. F. R. Gastrointestinal Manifestations of Food Allergy. Med. J. and Rec., 1925, 122, 271-275.
- 643. CARR, J. G. Hypersensitiveness to Milk Complicating the Treatment of Duodenal Ulcer. Med. Clin. North Amer., 1926, 9, 1409-1411.
- 644. Duke, W. W. Food Allergy as a Cause of Illness. J. Amer. Med. Assoc., 1923, 81, 886-889.
- 645. Duke, W. M. Mental and Neurologic Reactions of the Asthma Patient. J. Lab. and Clin. Med., 1927-1928, 13, 20-23.
- 646. GERRISH, F. H. Psychotherapeutics. Chap. II. Boston: Richard G. Badger, The Gorham Press, 1912.
- 647. MAISEL, F. E. The Asthma Problem. Med. J. and Rec., 1931, 134, 543-544.
- 648. Rowe, A. H. Food Allergy. Philadelphia: Lea and Febiger, 1931.
- 649. Rowe, A. H. Food Allergy in the Differential Diagnosis of Abdominal Symptoms. Amer. J. Med. Sci., 1932, 183, 529-537.
- 650. SHEARD, C., CAYLOR, H. D., and SCHLOTTHAUER, C. Photosensitization of Animals After the Ingestion of Buckwheat. J. Exp. Med., 1928, 47, 1013-1028.
- 651. Shannon, W. R. Neuropathic Manifestations in Infants and Children as a Result of Anaphylactic Reaction to Foods Contained in Their Dietary. Amer. J. Dis. Child., 1922, 24, 89-94.
- 652. Sherwood-Dunn, B. The Cause of Chronic Diseases, and Their Treatment by Entero-Antigens. *Internat. Clin.*, 1923, 4, 33rd series, 60-76.
- 653. Talbot, F. B. The Relation of Food Idiosyncrasies to the Diseases of Childhood. Boston Med. and Surg. J., 1918, 179, 285-288.
 - Also, see Nos. 273, 274, 275, 276, 279, 282, 283, 284, 285, 287, 288, 289, 290, 292, 298, 299, 305, 306, 308, 309, 311, 314, 316, 318, 319, 320, 321, 322, 325, 326, 328, 329, 330, 350, 352, 354, 363, 364, 365, 366, 371, 383, 384, 386, 387, 388, 395, 397, 398, 399, 545, 546, 639, and 662.

Sex Expression

- 654. Evans, H. M. Invariable Occurrence of Male Sterility with Dietaries Lacking Fat Soluble Vitamine E. *Proc. Nat. Acad. Sci.*, 1925, 11, 373-377.
- 655. Evans, H. M. The Effect of Inadequate Vitamin B upon Sexual Physiology in the Male. J. Nutrition, 1928, 1, 1-21.
- 656. MILES, W. R. The Sex Expression of Men Living on a Lowered Nutritional Level. J. Nerv. and Ment. Dis., 1919, 49, 208-224. Also, see Nos. 481, 502, 516, and 555.

Longevity

- 657. SHERMAN, H. C., and CAMPBELL, H. L. Further Experiments on the Influence of Food upon Longevity. J. Nutrition, 1930, 2, 415-417.
- 658. SHERMAN, H. C., and MacLeod, F. L. The Relation of Vitamin A to Growth, Reproduction and Longevity. J. Amer. Chem. Soc., 1925, 47, 1658-1662.
- 659. SLONAKER, J. R. The Effect of Different Per Cents of Protein in the Diet. VII. Life Span and Cause of Death. Amer. J. Physiol., 1931, 98, 266-275.

Hypertension

- 660. Jump, H. D. Essential Hypertension. Med. Clin. North Amer., 1924, 8, 763-770.
- 661. ROWLAND, V. C. The Dietetic Control of Some Forms of Hypertension and the Associated Gastrointestinal and Nervous Symptoms. Ann. Int. Med., 1932, 5, 971-981.

Meniere's Disease

- 662. Duke, W. W. Meniere's Syndrome Caused by Allergy. J. Amer. Med. Assoc., 1923, 81, 2179-2181.
- 663. KOPETZKY, S. J. The Meniere Symptom Complex. New York Med. J., 1913, 97, 1070-1076.
- 664. RICHEY, DEW. G. Oto-Rhino-Laryngology: Meatless Diet as a Therapeutic Measure in Meniere Disease. Amer. J. Med. Sci., 1930, 180, 588.

Beriberi

- 665. Funk, C. The Effect of a Diet of Polished Rice on the Nitrogen and Phosphorus of the Brain. J. Physiol., 1912, 44, 50-53.
- 666. LOVELACE, C. The Etiology of Beriberi. J. Amer. Med. Assoc., 1912, 59, 2134-2137.
- 667. VEDDER, E. B. Beriberi. New York: Wm. Wood and Co., 1913. Pp. 427.
- 668. ZIMMERMAN, H. M., and BURACK, E. Lesions of the Nervous System Resulting from Deficiency of the Vitamin B Complex. Arch. Path., 1932, 13, 207-232.

Also, see Nos. 136 and 436.

General

669. Fritz, M. F. The Field of Psychodietetics. Psychol. Clin., 1933, 22, 181-186.